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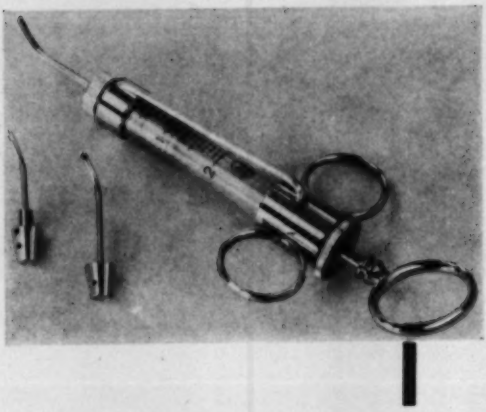

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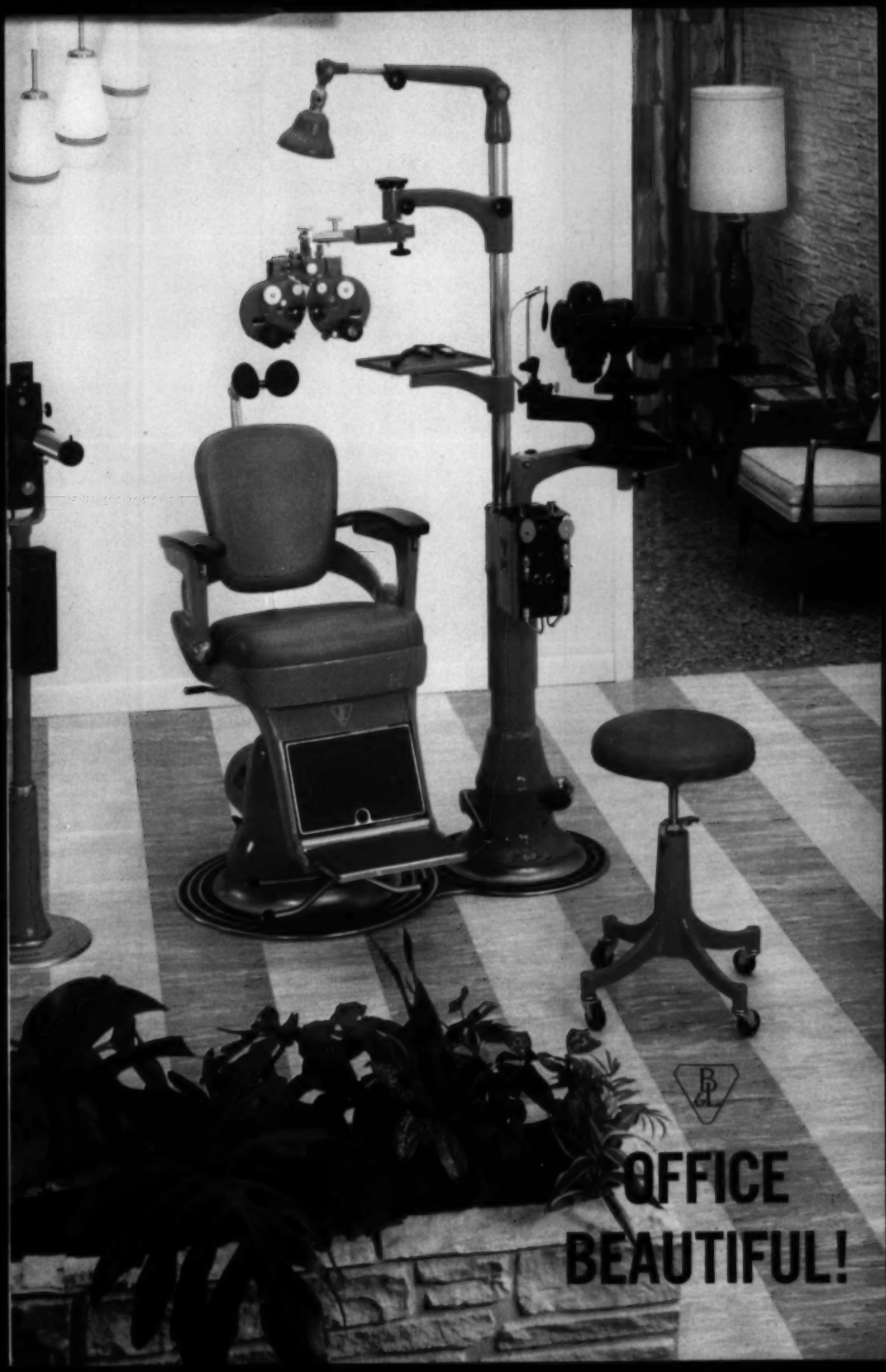
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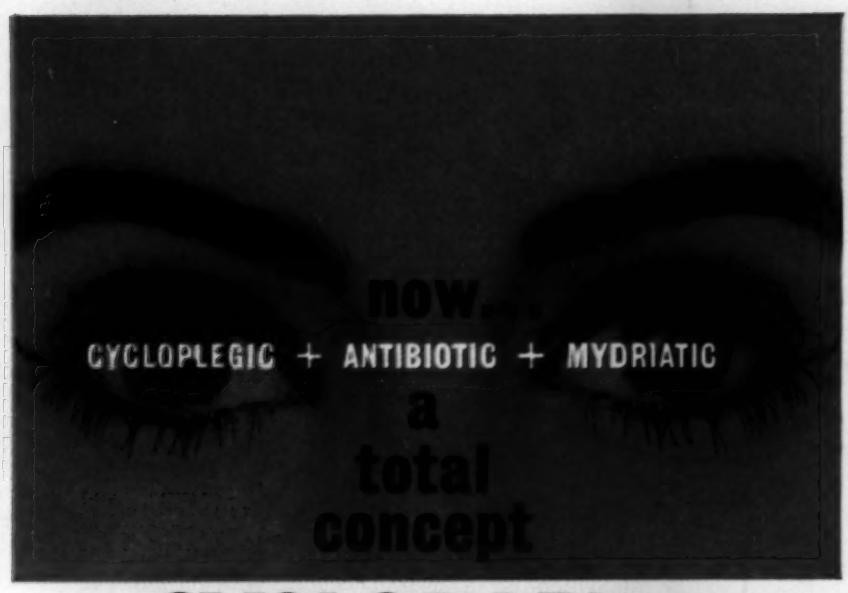

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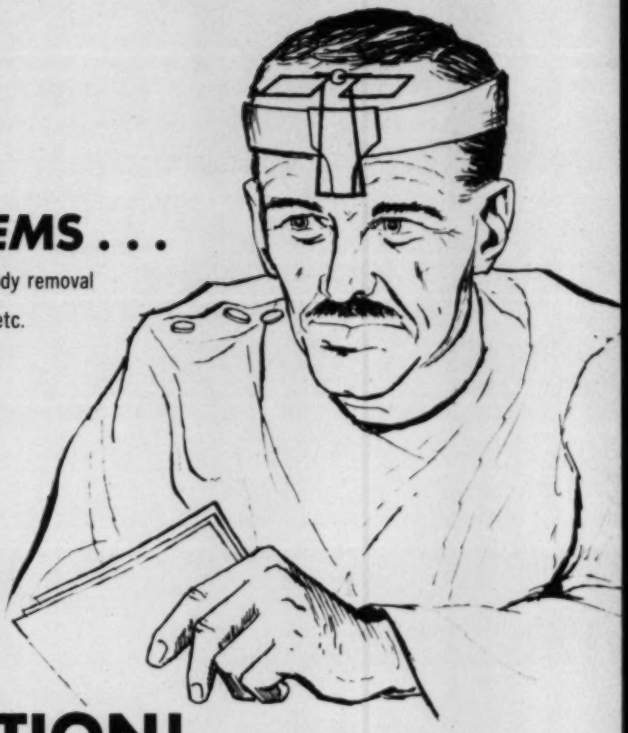

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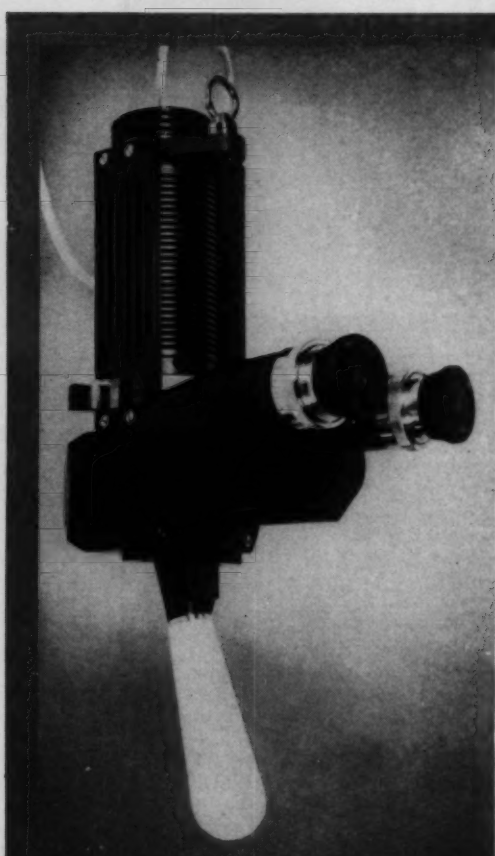

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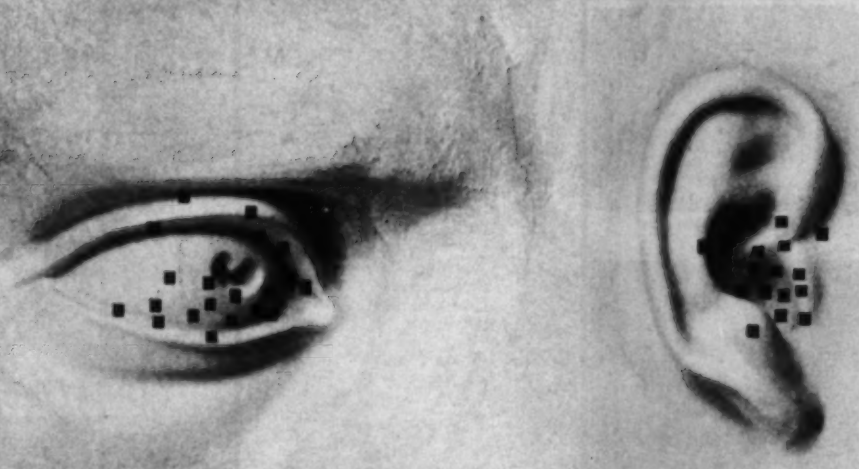

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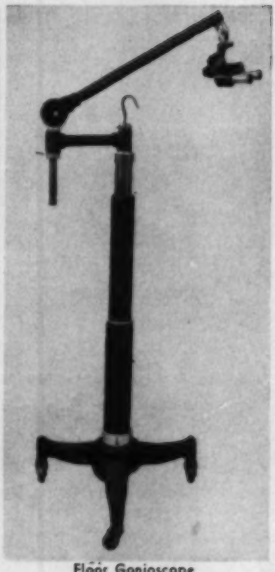

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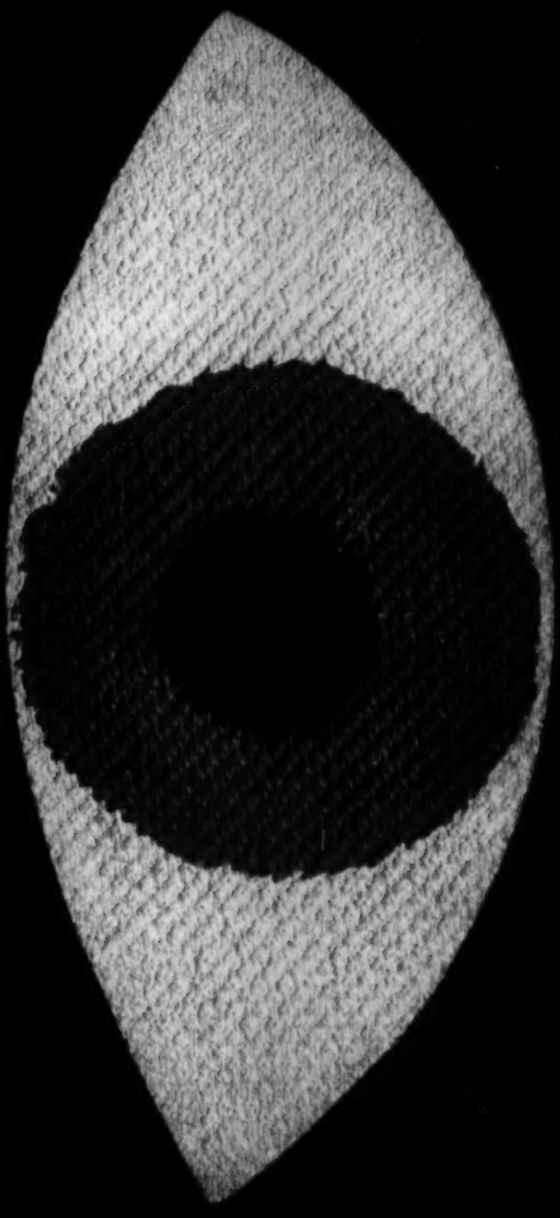
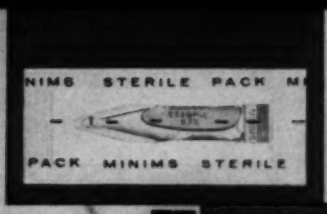
References: (1) Perkins, E. S.: **Practitioner 178**:575, 1957. (2) Queries and Minor Notes, **J.A.M.A. 161**:1032, 1956. (3) Smith, C. H.: **Eye, Ear, Nose & Throat Month. 34**:580, 1955. (4) Blakiston's New Gould Medical Dictionary, ed. 2, New York, McGraw-Hill Book Company, Inc., 1956, p. 945. (5) Ostler, H. B., & Braley, A. E.: **J. Iowa M. Soc. 44**:427, 1954.





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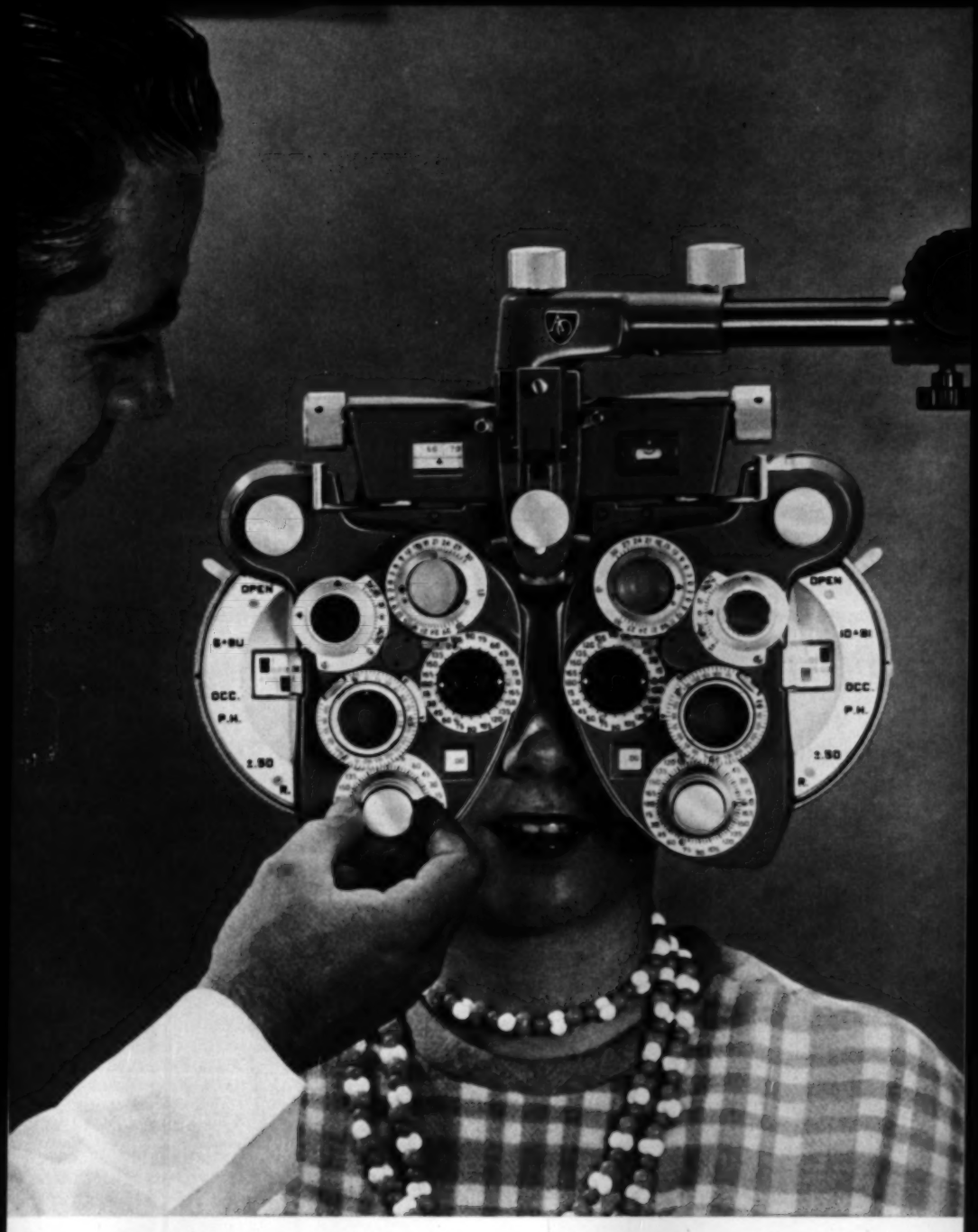

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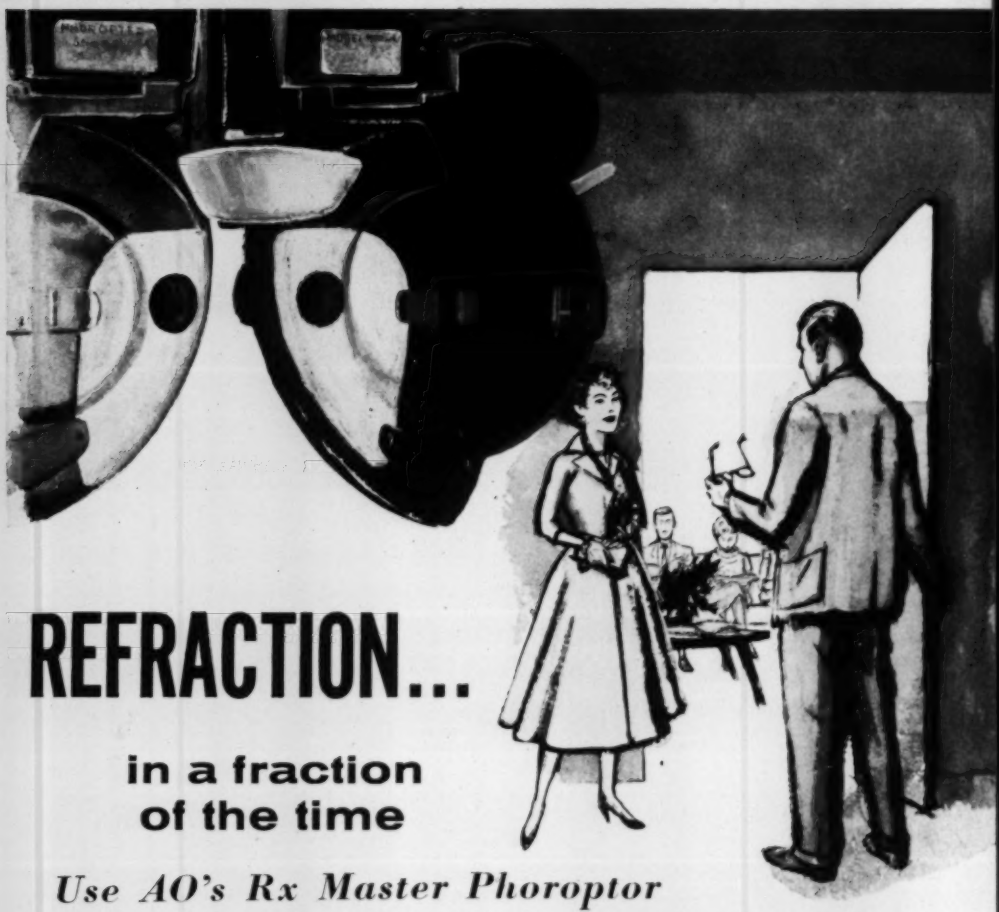

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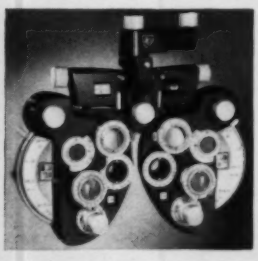

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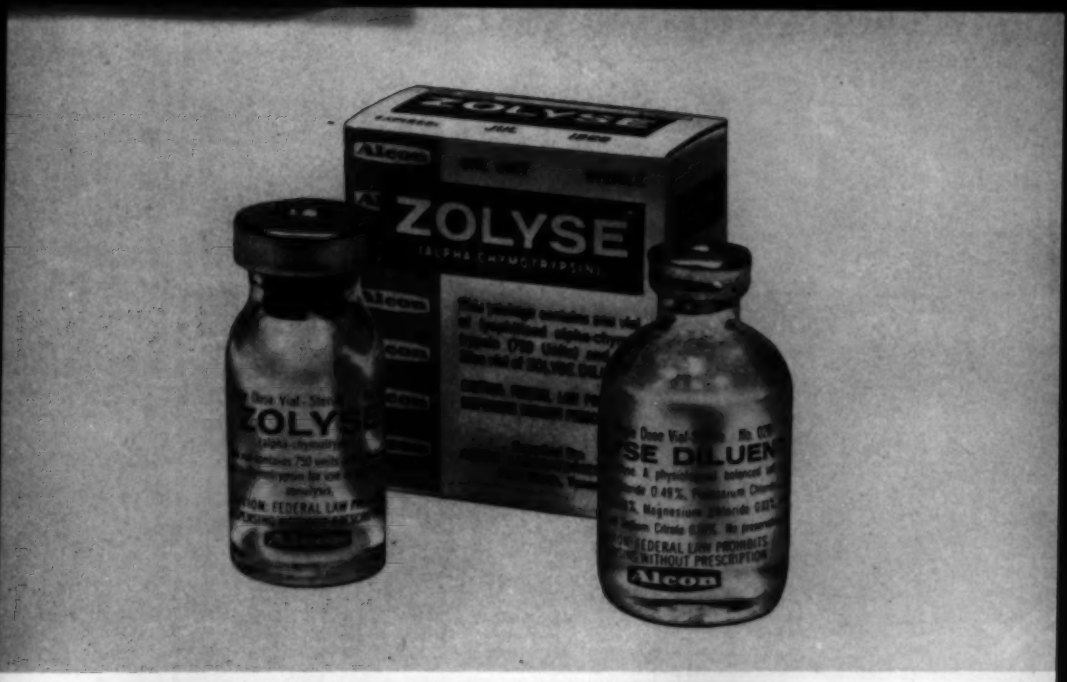

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[1] Girard, L. J., Dukes, C. D., and Fleming, T. C. Presented at the International Congress of Ophth., Brussels, Belgium, 1958.
[2] Kara, Gerald B., "The Use of Alpha-Chymotrypsin in Cataract Extraction," Research Report No. 10, Alcon Laboratories, Inc., 1959.
[3] Vail, D., et. al, Report: Committee on use of alpha-chymotrypsin in ophthalmology. Presented at the Sixty-fourth Annual Session, American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 11-16, 1959. (To be published)

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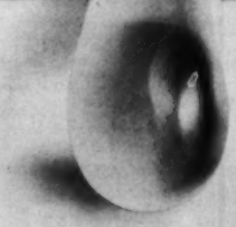

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1. Cogan, J. E. H.: Proc. Roy. Soc. Med. 51:927, 1958. 2. Jenkins, B. H.: J.M.A. Georgia 45:431, 1956. 3. Raiford, M. B.: J.M.A. Georgia 48:163, 1959. 4. Rizzuti, A. B.: A.M.A. Arch. Ophth. 61:135, 1959.

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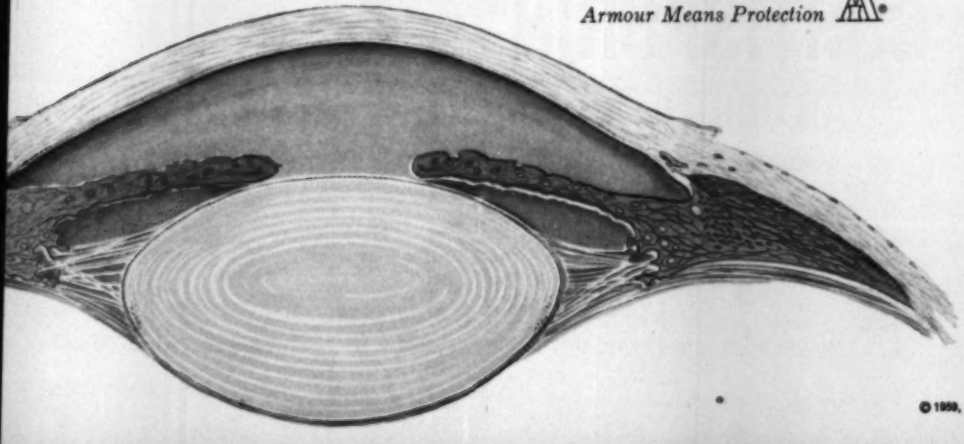

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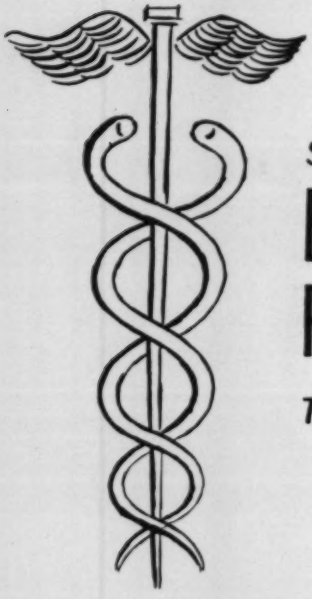

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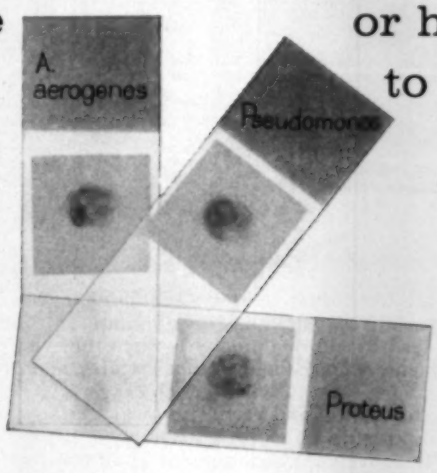

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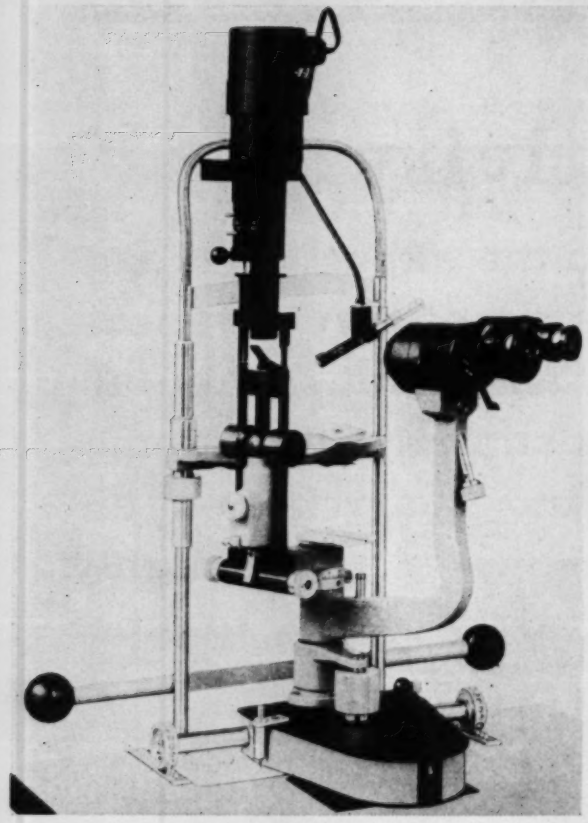

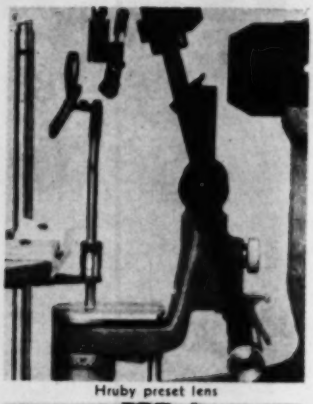

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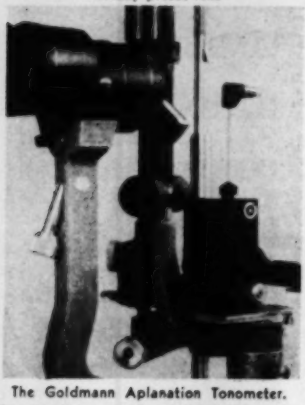

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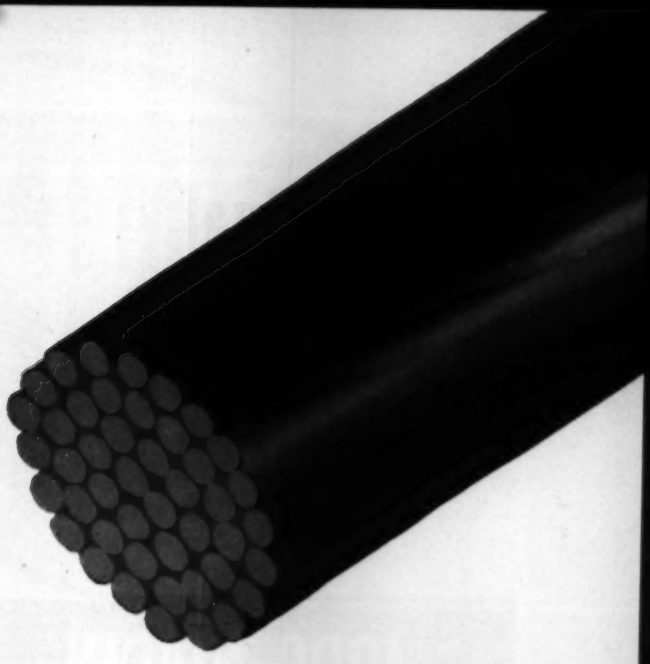

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1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.

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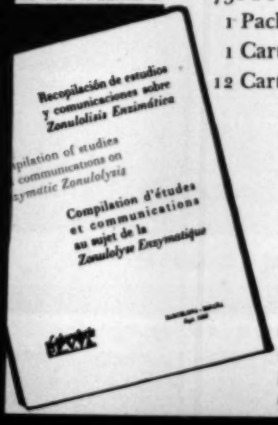
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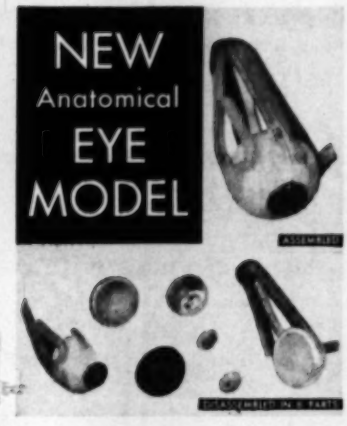

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References:

Beacher, L. Lester. "Photophobia," Chap. XVII in *Ocular Refraction and Diagnosis*. New York, 1931.

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Luckiesh, M. and Guth, S. K. "Brightness in Visual Field at Borderline between Comfort and Discomfort." *Illuminating Engineering*, Vol. 44 (November, 1949), pp. 650-667.

von Noorden, G. K. and Burian, H. M. "Visual Acuity in Normal and Amblyopic Patients under Reduced Illumination." *Archives of Ophthalmology*, Vol. 61, (April, 1959), pp. 533-535.

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examining each patient for the need for absorptive lenses.

A careful testing procedure eliminates uncertainty.

1. Learn all you can about the lighting conditions under which the patient does his daily work. Direct glare over the working area, harsh contrasts or bright spots from light sources within the field of vision may keep the patient from enjoying the full benefit of his correction. Just recently lighting engineers have revised upward their foot-candle recommendations for occupational visual tasks (see list of references). This suggests that light-sensitive patients will need correspondingly greater attention.

2. Relate above findings to the results of the refraction, to determine whether neutral absorption may be a desirable fourth component in the prescription. Pay particular attention to high minus corrections and cases of sub-normal vision.

3. Use the Soft-Lite Demonstrator over the patient's correction, with a reading card or chart. Ask if patient notices improvement in clarity or comfort of Soft-Lite shade 1 over white, and shade 2 over white. Often small improvements in acuity appear at this point. The test helps to identify light-sensitivity where it is one of a group of sub-threshold factors which together produce a complaint.

4. Discuss with the patient the function of neutral absorption in his prescription—its contribution to his comfort—and explain also the cosmetic features of absorptive lenses.

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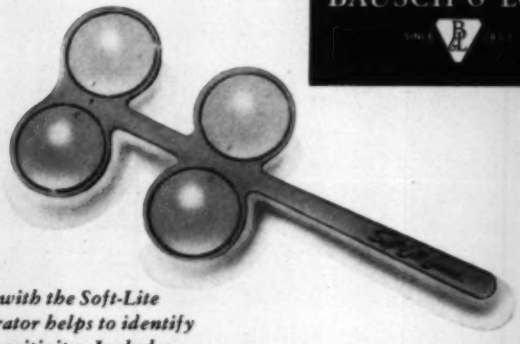

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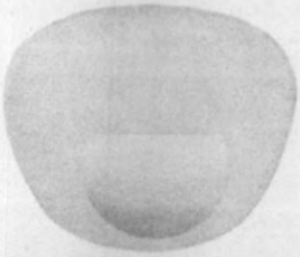

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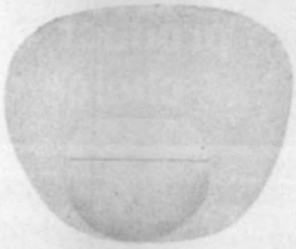

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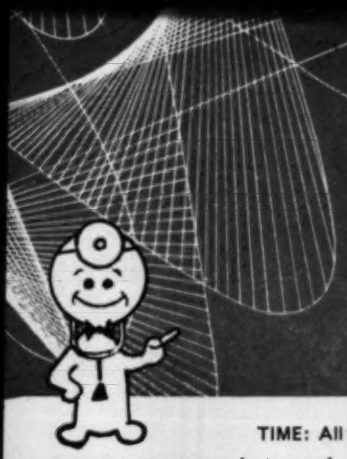

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Anatomy, embryology, and comparative ophthalmology; General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Tumors; Injuries; Congenital deformities, heredity; Hygiene, sociology, education, and history 390

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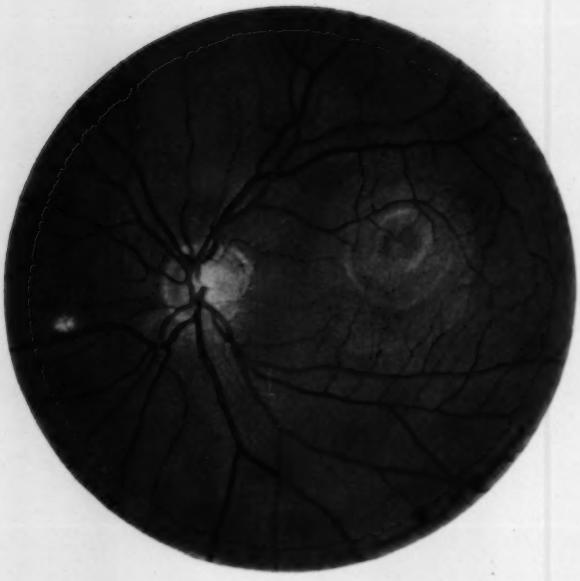

Fig. 11

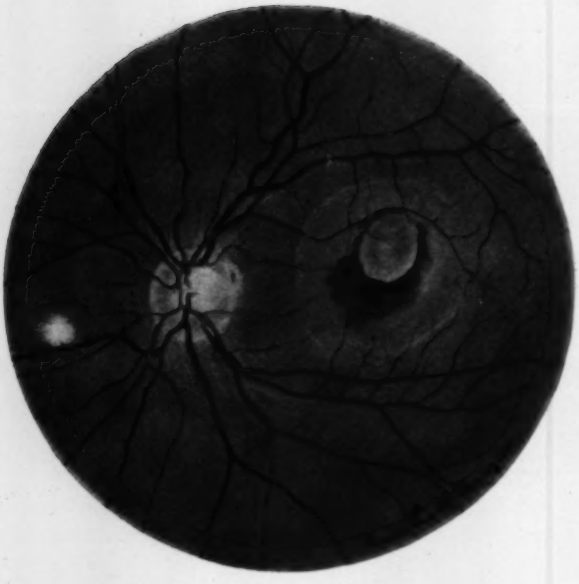

Fig. 12

Figs. 11 and 12 (Woods and Wahlen). The probable role of benign histoplasmosis in the etiology of granulomatous uveitis.
Fig. 11. Presumed histoplasmic chorioretinitis prior to diagnostic intracutaneous injection of histoplasmin (J.H.H. #824,987).
Fig. 12. Same case as in Figure 11, showing the focal reaction 48 hours after diagnostic injection of histoplasmin.

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VOLUME 49 FEBRUARY, 1960 NUMBER 2

THE PROBABLE ROLE OF BENIGN HISTOPLASMOSIS IN THE ETIOLOGY OF GRANULOMATOUS UVEITIS*

ALAN C. WOODS, M.D., AND HENRY E. WAHLEN, M.D.
Baltimore, Maryland

The purpose of this communication is to report the results of an exploratory study on the ambiguous question of the role of histoplasmosis in the etiology of uveitis. A brief summary of the present knowledge on systemic and ocular histoplasmosis is necessary to clarify the actual problem.

SYSTEMIC HISTOPLASMOSIS

The Histoplasma capsulatum is a small, gram-positive organism, about 3.0 to 5.0 micra in diameter with a round or crescentic central staining mass surrounded by a clear zone and a refractile capsule. It was first isolated by Darling,[1] in 1908, from the autopsy material of a supposed case of kala-azar. For a number of years it was classed as a protozoa, but in 1912 was correctly identified as a fungus with a mycelial and a yeast-like phase.

Until the introduction of histoplasmin as a diagnostic agent in 1941, human histoplasmosis was supposed to be a rare and usually fatal infection. Thus in 1940, Meleney[2] was able to collect only 32 cases, all except two having terminated in death. In 1945, Parsons and Zarafonetis[3] again reviewed the literature and assembled 71 cases, including seven of their own. Fifty-six of these had been fatal and the H. capsulatum had been recovered at autopsy from the lesions. In the 15 nonfatal cases the organism had been recovered from either tissue scrapings or the blood stream.

The symptomatology of these cases was most protean. In some the clinical picture was one of a systemic febrile disease, with various combinations of a septic temperature, generalized lymphoadenopathy, enlargement of the liver and spleen, and involvement of the bone marrow with a resultant leukopenia and anemia. In some cases the symptoms suggested Hodgkin's disease or lymphosarcoma, while in others the pulmonary changes predominated. There was also a mucocutaneous form with ulcerative lesions involving the skin, the naso-oral cavity, the gastro-intestinal tract, and so forth.

Histologically the systemic lesions of this fatal disease closely mimicked tuberculosis. There were tuberclelike nodules of epithelioid cells, often with giant cells, and usually with central caseation. The H. capsulatum was abundantly present in these lesions. It had a predeliction for the reticuloendothelial cells, and in the active stages of the disease was present in the phagocytes lining the sinusoids of the affected lymph glands, and in the large wandering cells of the blood stream.

The introduction of histoplasmin as a diagnostic agent in 1941 (Zarafonetis and Lindberg[4]; van Pernis et al.[5]) radically changed the currently held opinions on the nature of human histoplasmosis. Histoplasmin is a skin testing antigen prepared from the protein of H. capsulatum. With the single exception of occasional cross-reactions with Blastomyces dermatitides it is highly specific. Epidemiologic studies with this anti-

* From the Wilmer Ophthalmological Institute, Johns Hopkins University and Hospital. This work was supported in part by a special traineeship (BT 399) from the National Institute of Neurological Diseases and Blindness, Public Health Service.

gen, notably those of Palmer,[6] in 1945, showed that human histoplasmosis is usually a mild, asymptomatic, subclinical infection, is widely prevalent in certain states and relatively infrequent in others. In those states in which the frequency of positive reactions to histoplasmin is high, the incidence of pulmonary calcification is also high, and Parsons concluded that a large proportion of the pulmonary calcification in tuberculin-negative persons is due not to tuberculosis, but probably to histoplasmosis.

In the *Symposium on Histoplasmosis,* held at the National Institute of Health in 1948, Peterson[7] summed up the present views held by mycologists on human histoplasmosis, that is, that histoplasmosis is rare as a fatal disease in man, but as an unsuspected and asymptomatic infection is quite commonplace, especially in certain areas. The epicenter for this subclinical infection is at the confluence of the great rivers or in the mid-Mississippi valley. Histoplasmin sensitivity is an allergic state induced by histoplasmosis, is not a reflection of the status of the infection, and it may persist for years after the active stages of the disease have subsided. After the primary infection the parasite enters the blood stream and produces a widespread parasitization, especially of the reticulo-endothelial cells of the lungs, liver, and spleen. Small areas of pulmonary calcification are a usual complication of the disease. However, the parasite cannot be isolated from the sputum, because the small granulomatous lesions do not break down, and there are no parasites discharged into the bronchi. The parasite disappears early from the blood stream, and thereafter the diagnosis usually can only be made on the presence of an unexplained pulmonary calcification, the hypersensitivity to histoplasmin, or on some other such serologic or immunologic response.

Schwarz,[8] in 1953, taking exception to a statement made by Birge[9] the previous year, estimated that there were 20 million cases of the subclinical disease in the United States. Again in 1956,[10] he summed up the present views as being that asymptomatic histoplasmosis was a frequent disease, that a positive skin test to histoplasmin definitely indicated a prior infection with the H. capsulatum, but was no evidence the patient now had the clinical disease, and that the complement fixation reaction was of value only if it was positive and repeated tests showed a rising titer, in which case it indicated an active and possibly progressive disease.

Ocular histoplasmosis

There is only one case of ocular involvement reported in the fatal form of human histoplasmosis. This was reported by Reed[11] et al. in 1942. These authors described "small, white, irregular areas surrounded by hemorrhage . . . not unlike tubercles" in the fundus of a 38-year-old Negro who died of systemic histoplasmosis. The diagnosis was proven at autopsy, but unfortunately the eyes were not obtained, and the final proof of the histoplasmic etiology of these ocular lesions is lacking.

In 1949, Day[12] reported experiments in which the direct injection of H. capsulatum spores into the anterior chamber of rabbits' eyes produced a self-limited, nodular iritis of from two to three months' duration. In the early, active stages of this inflammation, the parasite could regularly be demonstrated in the sections, but as the granulomatous lesions became atrophic, they became scarce and finally could not be found. Day found that systemic injections of the spores, given four to six weeks prior to the anterior chamber injection, conferred on such rabbits a partial immunity to the ocular injection. One out of four rabbits given repeated intravenous injections of the living spores, developed a spontaneous granulomatous lesion in one eye, but examination of this eye one month later failed to demonstrate the organism.

The interest of ophthalmologists in histoplasmosis centers not on the acute, but on the benign form of the disease. Is benign histoplasmosis, like its counterpart acquired adult toxoplasmosis, an actual cause of endogenous

uveitis? Both are widespread, benign, asymptomatic and unsuspected infections. However, unlike the Toxoplasma gondii, the parasite of histoplasmosis has not been demonstrated in the lesions of an enucleated human eye. Nevertheless, there is some clinical and circumstantial evidence that a benign Histoplasma infection may be the actual cause of some cases of endogenous uveitis.

In 1948, small amounts of histoplasmin became available in the Wilmer Institute for investigative purposes. Using this material, Day[12] made intracutaneous tests on 118 unselected patients on the public wards of the Institute. Ninety-seven of these patients were entirely free of uveitis, and 29 of these gave positive reactions, an incidence of 30 percent. The remaining 21 patients had some form of uveitis, and in this group there were 14 positive reactors, an incidence of 67 percent. Four of these had pulmonary calcification and anergy to tuberculin.

In April, 1951, Krause[13] reported a patient with an atrophic, paracentral choroidal lesion with a small flame-shaped hemorrhage at its margin. This patient had pulmonary calcification, anergy to tuberculin, a strongly positive reaction to histoplasmin, a positive Histoplasma complement-fixation test, negative tests to other fungus antigens, and the medical survey had revealed no other cause to which the choroidal lesion could be attributed. Following the skin test with histoplasmin, the patient developed a focal hemorrhagic reaction around the old lesion. Efforts to isolate the H. capsulatum from the blood and bone marrow of this patient were fruitless. Krause believed the focal reaction in this patient was definite evidence of the histoplasmic etiology of the choroidal lesion.

In May, 1951, at the meeting of the Wilmer Resident's Association, one of us (A.C.W.) reported a patient who had a peculiar cystic lesion about one disc diameter in size near the macula, together with small, discrete focal scars scattered throughout the peripheral fundus. This patient was violently positive to histoplasmin (fig. 1), had pulmonary calcification, anergy to tuberculin, and the medical survey had disclosed no other cause which could be incriminated as the cause of the uveitis.

Fig. 1 (Woods and Wahlen). Positive reaction to histoplasmin. (J.H.H. #565530.)

For the next two years, intracutaneous tests with histoplasmin were done in the Wilmer Institute on uveitis patients who had pulmonary calcification, and were tuberculin negative. In 1953, the uveitis survey was amplified to include routine histoplasmin tests in all patients, regardless of the granulomatous or nongranulomatous nature of their uveitis. Except for occasional lapses when the supply of histoplasmin was exhausted, and omissions due to oversight or inability to complete the survey, this routine has been consistently followed to date.

During this five-year period, a number of patients in the granulomatous group were encountered who showed positive histoplasmin tests, pulmonary calcification, and anergy to tuberculin, but, more often than not, other positive findings clouded the picture and no special attention was given to histoplasmosis as an important cause of uveitis. However, in the fall of 1958, three patients were seen in rapid succession who not only fulfilled the above criteria, but also had fundus pictures reminiscent of that shown by the patient with histoplasmosis reported in 1951. The observations on these patients suggested that it might well be profitable to review and ana-

TABLE 1

GEOGRAPHIC DISTRIBUTION, OVERALL TOTALS AND PERCENTAGE INCIDENCE OF POSITIVE AND NEGATIVE REACTORS TO HISTOPLASMIN IN 186 PATIENTS WITH GRANULOMATOUS UVEITIS

	Mid-Eastern States		Mid-Central States		Southern States	Scattering	Overall Totals and Percentage Incidence
	Maryland	Others	Ohio	Others			
Total No.	40	58	19	19	30	20	186
Positive Reactors	16 (40%)	8 (15%)	14 (70%)	10 (53%)	10 (33%)	4 (20%)	62 (33%)
Negative Reactors	24 (60%)	50 (85%)	5 (30%)	9 (47%)	20 (67%)	16 (80%)	124 (67%)
Controls (Palmer 1946)	31% pos. 69% neg.	13% pos. 87% neg.	60% pos. 40% neg.	52% pos. 48% neg.	6% pos. 94% neg.	— —	21% pos. 79% neg. (8,141 tests)

lyze the material on histoplasmosis which had been collected over these years.

MATERIAL

The material on which this study is based are the histories of 186 patients with granulomatous uveitis and 107 patients with nongranulomatous uveitis. All of these patients have been seen since 1951, and all have had complete medical surveys, including histoplasmin tests, to determine, if possible, the cause of their uveitis. The histories of patients on whom the survey was incomplete or on whom the results of the histoplasmin tests were equivocal, have been excluded.

The histories of all these patients were first reviewed and the incidence of positive reactions to histoplasmin was determined. The histories of the positive reactors were then further analyzed, the positive findings disclosed by the medical survey were charted, the details of the ocular lesions were recorded, and in the light of this information, the possible relation of benign histoplasmosis to the uveitis was evaluated. The results of this study are as follows:

RESULTS

1. INCIDENCE OF HISTOPLASMIN SENSITIVITY

In the 186 patients with granulomatous uveitis, there were 62 positive reactors to histoplasmin and 124 negative reactors. In the 107 patients with nongranulomatous uveitis, there were 16 positive reactors and 91 negative reactors. This is an incidence respectively of 33 percent and 15 percent in the two series. However, before any conclusions can be drawn from these figures, the geographic distribution of the patients must be considered.

It is well known that in certain states, particularly the eastern midcentral ones, the incidence of positive histoplasmin reactors is extremely high, while in other sectors of the country it is very low. In 1946, Palmer[14] published a report showing the incidence of positive reactions to histoplasmin in 8,141 student nurses with lifetime residence in different sections of the United States. This report clearly illustrates these wide differences.

In Table 1 are shown the geographic distribution and percentage incidence of positive and negative histoplasmin reactions for 186 patients with granulomatous uveitis. In Table 2 these same factors for 107 patients with nongranulomatous uveitis are similarly shown. The first three groups in both tables are comprised of patients residing in the same states. The fourth group includes all patients coming from other localities. Also shown in these tables are figures taken from Palmer's

TABLE 2

GEOGRAPHIC DISTRIBUTION, OVERALL TOTALS AND PERCENTAGE INCIDENCE OF POSITIVE AND NEGATIVE REACTORS TO HISTOPLASMIN IN 107 PATIENTS WITH NONGRANULOMATOUS UVEITIS

	Mid-Eastern States		Mid-Central States		Southern States	Scattering	Overall Totals and Percentage Incidence
	Maryland	Others	Ohio	Others			
Total No.	10	44	5	10	15	23	107
Positive Reactors	2 (20%)	4 (9%)	3 (60%)	5 (50%)	1 (7%)	1 (4%)	16 (15%)
Negative Reactors	8 (80%)	40 (91%)	2 (40%)	5 (50%)	14 (93%)	22 (96%)	91 (85%)

report showing the incidence of positive reactions among presumably normal nurses residing in these same states, together with the over-all incidence found by him.

When due allowances are made for the smaller number of patients with nongranulomatous uveitis residing in the midcentral states, and the incidence of positive reactors in the two series is compared, it is immediately evident that the incidence of positive reactors (33 percent) in the patients with granulomatous uveitis is approximately twice that observed in patients with nongranulomatous uveitis (15 percent). When these figures are subjected to statistical analysis, the probability that this difference is due to chance is less than 0.01.

The incidence of positive reactors among patients with granulomatous uveitis is definitely higher than that found by Palmer, while the incidence of positive reactors among patients with nongranulomatous uveitis is approximately that found by him for normal individuals. These figures are of statistical significance.

2. CLASSIFICATION OF POSITIVE REACTORS IN GRANULOMATOUS UVEITIS PATIENTS

When the case histories of the positive histoplasmin reactors among the granulomatous uveitis patients were reviewed in the light of the criteria employed for a diagnosis of histoplasmosis and the positive findings disclosed by the medical survey, it was immediately evident they could be classified in six distinct groups. These were as follows:

Group I. Patients who fulfilled the usual criteria for a diagnosis of systemic histoplasmosis, and in whom the medical survey had disclosed no other cause to which the uveitis could be attributed. The criteria for the diagnosis of histoplasmosis were:

A. Positive reactions of at least 15 by 15 mm. erythema with central induration to an intracutaneous injection of 0.1 cc. of a 1:100 dilution of histoplasmin.

B. Radiographic evidence of pulmonary calcification.

C. Complete anergy to an intracutaneous injection of 0.1 mg. of old tuberculin.

Group II. Patients who were hypersensitive to histoplasmin, were nonreactive to tuberculin and in whom the medical survey had revealed no cause other than histoplasmosis to which the uveitis could be attributed. These patients, however, did not have radiographic evidence of pulmonary calcification.

Group III. Patients who were hypersensitive to histoplasmin, had pulmonary calcification, were anergic to tuberculin, but in whom the medical survey had disclosed evidence of other systemic granulomatous disease to which the uveitis might conceivably be attributed.

Group IV. Patients who were hypersensitive to histoplasmin, had pulmonary calcification, but in whom the tuberculin reaction was positive. The medical survey had dis-

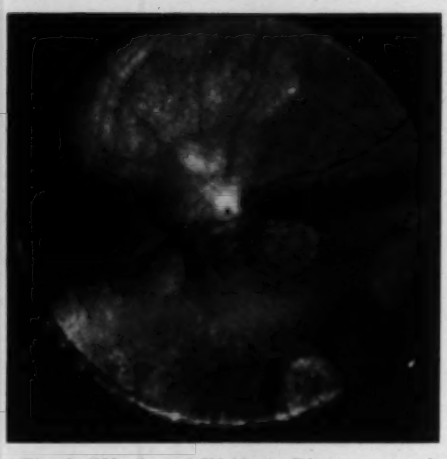

Fig. 2 (Woods and Wahlen). Discrete, sparsely pigmented lesions in peripheral fundus. (J.H.H. #565530.)

closed no evidence of systemic granulomatous disease other than histoplasmosis and tuberculosis to which the uveitis might be attributed.

Group V. Patients hypersensitive to histoplasmin and with pulmonary calcification, but who reacted to tuberculin and also showed evidences of a systemic granulomatous disease other than histoplasmosis and tuberculosis.

Group VI. Patients who were hypersensitive to histoplasmin, but who showed no other evidence of the disease. If pulmonary calcification was present, it was believed to be due to an old tuberculosis. In addition, the patients in this group usually showed evidence of other systemic granulomatous disease.

The ocular pictures in these six groups were as follows:

3. Ocular lesions in granulomatous uveitis patients hypersensitive to histoplasmin

Group I. The nine patients in this group all were histoplasmin positive, had pulmonary calcification, were nonreactive to tuberculin, and in none had the medical survey revealed any other cause to which the uveitis could be attributed. The character of the ocular lesion in all nine patients was remarkably similar and consistent. In five cases the lesions were bilateral, and in four unilateral.

In no case was there any involvement of the anterior uvea. As a rule the vitreous was clear; in a few cases only there were a few discrete opacities. Thirteen of the 14 involved eyes had both peripheral and central lesions. One patient apparently had the central lesion only, there being no note in the history of any peripheral lesions.

Fig. 3 (Woods and Wahlen). Unpigmented, small peripheral lesions in nasal fundus. (J.H.H. #838540.)

Fig. 4 (Woods and Wahlen). Discrete lesions in peripheral fundus. (J.H.H. #829601.)

The peripheral lesions consisted of discrete, focal spots of atrophic chorioretinitis. These were usually about one-third disc diameter or less in size, were often somewhat yellowish in color, and were either depigmented or sparsely pigmented (figs. 2, 3, and 4). In one case, that of a 12-year-old boy, the scars were more diffuse and more heavily pigmented.

The central lesions were either directly in the macula or closely contiguous to it. In the early cases they consisted of a small subretinal cyst with a surrounding halo of edema or subretinal serous exudate (fig. 5). In older cases the cystic lesions were larger, a disc diameter or more in size, again surrounded by an area of subretinal effusion, the whole macula being elevated (fig. 6). Secondary hemorrhage, occurring a few weeks or even months after the primary cystic lesion was first noted, was a complication in five of the 10 cases. These began as a zone of deep congestion and progressed to an actual hemorrhage around the central cystic lesion (fig. 7).

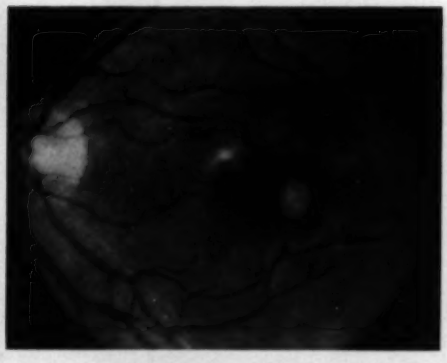

Fig. 5 (Woods and Wahlen). Early central, cystic lesion. (J.H.H. #829962.)

In the final atrophic stage, after the serous effusion had subsided, the central lesion appeared either as an area of superficial retinal degeneration (fig. 8), as a circular well-outlined glial scar (fig. 9), or as an irregular mass of heaped-up, pigmented glial tissue, indistinguishable from a Junius-Kuhnt disciform macular degeneration, which in truth it actually was (fig. 10). In one case the end-picture was a hole in the macula with a little surrounding degeneration.

In three of the bilateral cases, one eye showed the peripheral scars with a central Junius-Kuhnt degeneration, while the second eye showed the peripheral scars and an active central cystic lesion. In the fourth case, both eyes showed the peripheral scars and active cystic central lesions. The last bilateral case showed the peripheral scars and a subsiding, hemorrhagic cystic lesion in one eye, while the second eye showed the same type of peripheral scars and a very early cystic lesion without hemorrhage (fig. 11). In this patient, beginning 24 hours after a diagnos-

Fig. 6 (Woods and Wahlen). More advanced central lesion, without hemorrhage. (J.H.H. #824987.)

tic intracutaneous injection of histoplasmin, the second eye suddenly developed an acute hemorrhagic reaction around the central lesion, and in the nasal fundus there was a sudden reactivation of a small lesion which previously had appeared entirely inactive (fig. 12). This reactivated lesion appeared as a soft, round exudate, almost exactly similar in appearance to the miliary choroidal tubercles which so often occur in the terminal stages of a tuberculous meningitis.

Three of the unilateral cases showed the characteristic combination of atrophic peripheral scars together with early, central, cystic lesions. In the fourth case, a patient who has disappeared from observation, only the central cystic lesion was described. Whether the peripheral lesions were present and were missed or ignored by the examiner, or whether they were truly absent, is an open question.

Group II. The two patients in this group were histoplasmin positive and tuberculin negative, but they did not have pulmonary calcification or other lung changes. The survey had revealed no evidence of granulomatous disease other than histoplasmosis. One of these patients had in one eye an atrophic central lesion together with the characteristic peripheral scars, while the fellow eye showed an acute hemorrhagic cystic foveal lesion together with the peripheral scars. The second patient had the typical peripheral scars in one eye, but the macula had been spared. The fellow eye had the same type of peripheral scarring together with a cystic lesion in the macula surrounded by a zone of hemorrhage. In view of the similarity of these ocular lesions to those of the Group I patients, and the absence of any other etiologic factor, the uveitis was attributed to a histoplasmic infection which had spared the lungs and the mediastinum.

Group III. This group consisted of six patients all of whom were histoplasmin positive, tuberculin negative, had pulmonary calcification, and in addition had positive toxoplasmin skin tests and strongly positive

Fig. 7 (Woods and Wahlen). Central cystic lesion with surrounding zone of hemorrhage. (J.H.H. #665137.)

Fig. 8 (Woods and Wahlen). Retinal degeneration after subsidence of cystic lesion. (J.H.H. #838548.)

Sabin-Feldman dye tests. The question was the differential diagnosis between a histoplasmic and a toxoplasmic etiology.

In the first four cases, there was no difficulty. The ocular lesions were all either heavy, circumscribed, pigmented glial scars, or an exudative focal chorioretinitis. There was no anterior segment involvement. These were all classed as of toxoplasmic etiology.

The last two cases were quite different. In the first one the patient developed a metamorphopsia in April, 1957. At this time, a cystic lesion in the macula was discovered, but the chest plate was negative and the patient was insensitive to tuberculin. No other examinations were made. Several months later she developed a hemorrhage around this central lesion. This absorbed, leaving a circular central scar. In January, 1959, on account of a pregnancy, she was referred for a diagnostic study. A histoplasmin test was now done and was strongly positive. A new chest plate was made and compared to the older one. Areas of pulmonary calcification, which had developed between the times the chest plates were taken, were now found. The tuberculin tests were still negative, but the toxoplasmin skin test and the dye test were both positive, the latter in high titer. Examination of the peripheral fundus dis-

Fig. 9 (Woods and Wahlen). Central chorioretinal degeneration after subsidence of central cystic lesion. (J.H.H. #665137.)

Fig. 10 (Woods and Wahlen). Junius-Kuhnt type of disciform macular degeneration resulting from central cystic lesions. (J.H.H. #775938.)

closed several old, small, discrete, unpigmented scars. Despite the positive tests for toxoplasmosis, it was believed the ocular picture and the sequence of the symptomatology justified the tentative diagnosis of a histoplasmic etiology.

The last patient in this group had the typical, small, peripheral lesions in both eyes, the macula being spared in one, while the other had an early, fresh, central, cystic lesion. Since these fundus lesions were totally unlike those usually seen in toxoplasmosis, and were exactly similar to those attributed to histoplasmosis in the other groups, it was felt the latter etiologic diagnosis was justified.

Group IV. The 10 patients in this group were histoplasmin positive, had pulmonary calcification, and reacted to various strengths of tuberculin. In none had the medical survey disclosed any evidence of other systemic granulomatous disease. The question therefore was the differential diagnosis between tuberculosis and histoplasmosis as the etiologic factor for the uveitis.

Two of these patients had a generalized granulomatous uveitis with mutton-fat keratic deposits, and one had a massive exudative choroiditis. On the strength of these typical pictures, these three cases were classified as of tuberculous etiology. In two other patients the picture was so confused that no decision appeared justified, and both were classified as of undetermined etiology.

The other five cases were classified as being probably due to histoplasmosis. In three the uveitis was bilateral and in two unilateral. The first of the bilateral cases had the usual peripheral scars in both eyes, with an atrophic lesion in one macula and a fresh central, cystic hemorrhagic lesion in the other. The second case showed bilateral peripheral lesions, but in one eye the macula was spared, while in the other there was an early cystic lesion, without hemorrhage, contiguous to the fovea.

The third bilateral case was most interesting. There was an active, granulomatous iritis, with a number of small, whitish nodules around the ciliary border of the iris in the right and a few similar iridic nodules in the left eye. There were keratic deposits of the epithelioid cell type in both eyes together with early lenticular changes. With indirect ophthalmoscopy a few scattered, atrophic yellowish spots could be made out in the periphery of both eyes, but the foveas could not be visualized. There was pulmonary calcification, violent hypersensitivity to histoplasmin, and a plus-minus reaction to the large dose of 0.1 mg. of tuberculin. This patient received adrenal steroid treatment in the Barnes Hospital in St. Louis, the iritis subsided, and both eyes were successfully operated for cataracts by Dr. Becker. Ophthalmoscopic examination after operation revealed the bilateral central lesions one of

which is illustrated in Figure 13.

The two patients with unilateral involvement only showed the characteristic peripheral scars together with central cystic lesions surrounded by a zone of active hemorrhage.

Group V. The 12 patients in this group all had the complicated picture of histoplasmin sensitivity, pulmonary calcification, hypersensitivity to tuberculin, and positive skin and dye tests for toxoplasmosis. The question was one of differential etiologic diagnosis.

Two of these patients, both with a generalized uveitis, responded dramatically to antitubercular therapy, and so were classified as of tuberculous etiology. Two others with large areas of active or atrophic focal choroiditis, and one patient with an active choroiditis and a "spill-over" to the anterior uvea, were classified as toxoplasmosis. In six patients a differential diagnosis could not be made and these were classified as of undetermined etiology. In only two of these was histoplasmosis even remotely considered as the etiologic factor.

The last patient in this group was tentatively classed as a case of histoplasmic uveitis. One eye showed the peripheral scars and a Junius-Kuhnt disciform degeneration at the macula. The second eye showed only the peripheral scars, the macula being spared. This patient lived on the same farm with a patient in Group IV, who had the typical picture of an early central lesion and peripheral scarring. On account of this geographic proximity, and the almost typical nature of the ocular picture, this tentative diagnosis appeared justified.

Group VI. This was a heterogenous collection of 23 patients with various forms of granulomatous uveitis, almost the only common denominator being the histoplasmin sensitivity. In three of these patients the ocular lesions were somewhat suggestive of those attributed to histoplasmosis in the preceding groups, but were not sufficiently typical to warrant such a diagnosis. Only three of these patients had pulmonary calcification, and in two this was believed due to tuberculosis and in one to sarcoidosis. Of the 20 patients with negative chest plates, 10 were tuberculin sensitive and 10 were tuberculin negative. Nineteen patients had evidences of one or more systemic granulomatous diseases—toxoplasmosis, tuberculosis, syphilis, sarcoidosis, and, in one patient, sympathetic ophthalmia. In four patients the medical survey had disclosed no evidence of any systemic granulomatous disease. Eleven had generalized uveitis, and 12 had various types of choroiditis. In 12 the assembled evidence appeared sufficient to warrant a diagnosis of one of the conventional etiologies. In 11 no definite diagnosis could be made.

Fig. 13 (Woods and Wahlen). Atrophic central lesion in presumed histoplasmic chorioretinitis—first seen after extraction of cataract. (J.H.H. #666705.)

Summary of positive histoplasmin reactors with granulomatous uveitis.

In 19 of the 62 patients in these six groups representing a total of 31 involved eyes, the uveitis was believed to be related to a benign systemic histoplasmosis. In only one patient were the anterior uveas involved. The posterior uveas were involved in all. In 20 of the 31 eyes, the fundus lesions consisted of a combination of atrophic, discrete, peripheral scars and an active, central, cystic lesion. In six eyes both the peripheral and the central lesions were atrophic. In four eyes only peripheral lesions were present, the maculas

TABLE 3

SUMMARY OF OCULAR PICTURE, MEDICAL FINDINGS, AND ETIOLOGIC DIAGNOSIS IN 43 PATIENTS WITH GRANULOMATOUS UVEITIS OF CONVENTIONAL OR UNDETERMINED ETIOLOGY BUT WITH POSITIVE HISTOPLASMIN REACTIONS

Group	No. of Patients	Ocular Picture				Medical Findings				Etiologic Diagnosis			
		Anterior Uveitis	Choroiditis		Peripheral or Central	Pulmon. Calcification	Chest Plate Negative	Reaction to Tuberculin	Other Granulomatous Disease	Tuberculosis	Toxoplasmosis	Other Granulomatous Disease	Undetermined
			Focal	Diffuse or Disseminated									
III	4	0	3	1	0	4	0	all neg.	4 (toxo.)	0	4	0	0
IV	5	2	3	0	0	5	0	all pos.	5 (Tbc.)	3	0	0	2
V	11	4	3	1	3	11	0	all pos.	11 (Tbc. toxo.)	3	2	0	6
VI	23	11	4	5	3	3	20	14 pos. 9 neg.	19 (Tbc. toxo. scattering)	2	6	4	11
	43	17	13	7	6	22	20	30 pos. 13 neg.	39*	8	12	4	19

* In four patients the surveys disclosed no evidence of any systemic granulomatous disease.

being spared. In one eye only the active, central lesion was noted.

In the eyes with the active lesions, it appeared that the foveal or perifoveal involvement had occurred after the peripheral lesions had become atrophic, but there was no way of determining how long the interval between them had been. The central lesions began as small cystic changes which usually progressed, and in about one half the cases were complicated by a later, secondary hemorrhage which had a tendency to surround the primary cystic lesion. The healed central lesions showed either various degrees of chorioretinal degeneration or a type of pigmented gliosis which was indistinguishable from the usual Junius-Kuhnt disciform macular degeneration.

The ocular changes and the essential findings revealed by the medical survey in the 43 positive reactors in whom the uveitis was not regarded as of histoplasmic etiology, are summarized in Table 3. In 24 of these patients, despite the positive histoplasmin tests, the over-all evidence was sufficiently strong to permit a diagnosis of one of the recognized, conventional etiologies with a reasonable assurance of accuracy. Nineteen cases were classified as of undetermined etiology. In three of these latter, the ocular lesions were somewhat suggestive of those in which a diagnosis of histoplasmic etiology had been made, but in no case was such a tentative diagnosis justified. In seven instances the diagnosis appeared to lie between tuberculosis and toxoplasmosis, and in five others, while there was some evidence of other systemic granulomatous disease, no plausible etiologic diagnosis was possible. In the remaining four cases the medical survey had disclosed no evidence of any systemic granulomatous disease.

It is of course quite possible that the uveitis in some of these cases listed as of undetermined etiology, and indeed in some of those classified as of conventional etiology, may in truth have been related to the Histoplasma infection. However, it was impossible to assume such a relationship on the basis of nothing more than a positive reaction to histoplasmin.

4. POSITIVE HISTOPLAMIN REACTORS IN PATIENTS WITH NONGRANULOMATOUS UVEITIS.

There were 16 positive reactors to histoplasmin in the 107 patients with nongranulomatous uveitis. This number is so small that it was fruitless to attempt to divide them into different groups. In only one instance did the assembled evidence suggest an etiologic diagnosis of histoplasmosis. While eight of these

positive reactors had pulmonary calcification, only two of them had anergy to tuberculin. These two patients both had a marked hypersensitivity to streptococci, and their uveitis appeared related to this bacterial sensitivity. In 13 other positive reactors, all with recurrent iridocyclitis, there was no reason to suspect histoplasmosis as the etiologic factor, and there was abundant reason to incriminate bacterial hypersensitivity and often focal infection in all of them.

The one exception was a 48-year-old woman (J.H.H. history #729637) who had suffered repeated attacks of bilateral nongranulomatous iridocyclitis over a two-year period. Examination of her eyes showed the usual residua of such iridic inflammation, and in addition there were small, discrete, yellowish, nonpigmented scars scattered over the peripheral fundi, similar to those observed in patients with a granulomatous uveitis of presumed histoplasmic etiology. There was no macular involvement in either eye. No bacterial hypersensitivity could be demonstrated and the survey had revealed no focus of infection, or other conventional cause, to explain the iridocyclitis. The patient was violently sensitive to histoplasmin and was completely anergic to tuberculin, but there was no pulmonary calcification. It appeared quite possible that in this patient the recurrent iridocyclitis might be related to a hypersensitivity to histoplasmin which had affected especially the anterior uvea. This patient and the one with a nodular iritis (in Group IV of the granulomatous uveitis series) are the only two instances encountered in the entire study in which there was reason to suspect a relationship between the histoplasmic infection and an anterior uveitis.

Pathogenesis

Any explanation of these ocular lesions as being due to a systemic histoplasmosis must take into account the following facts.

A. While the trilogy of histoplasmin sensitivity, pulmonary calcification, and anergy to tuberculin are indicative of a former infection with the H. capsulatum, they do not certify that the patient now has the active disease. An active, progressive infection should be manifested by the development of new granulomatous lesions, by the lighting up of old ones, and an increasing immunologic response by the host. The patients with the presumed histoplasmic uveitis here reported were entirely asymptomatic except for their eyes. Efforts to demonstrate a rising complement-fixation titer in them were unsuccessful. While the complement-fixation reaction is a notoriously unsatisfactory tool in the diagnosis of ocular infections, and it is probable that a small focus of active inflammation in an eye alone does not exert sufficient antigenic activity to influence the production of a skin sensitivity or of complement-fixing antibodies to any appreciable degree, nevertheless, the fact remains that while many of these patients did show a complement-fixation reaction positive in low titers, such positive reactions were variable, inconstant, and unpredictable, and in no case was a definitely rising titer demonstrated. Possibly if an agglutination test such as that suggested by Saslaw[15] and employed by Salvin and Furcolow,[16] a dye test such as is used successfully in toxoplasmosis, or an hemo-agglutination reaction, could be adapted for the diagnosis of histoplasmosis, some change in the immunologic or serologic response of these patients might be detected. Until that is done, it must be admitted that the present evidence indicates that in these patients with a presumed histoplasmic uveitis, any systemic infection is either latent, quiescent, or healed.

B. The H. capsulatum has never been demonstrated in any lesions in a human eye. Day's experimental studies showed that in the early stages of the disease, the spores could be demonstrated in the granulomatous lesions which followed the direct injection of the spores into the eye. However, as the lesions became older and atrophic, the parasite could no longer be found in the histologic sections, although in one such case their presence was demonstrated by culture

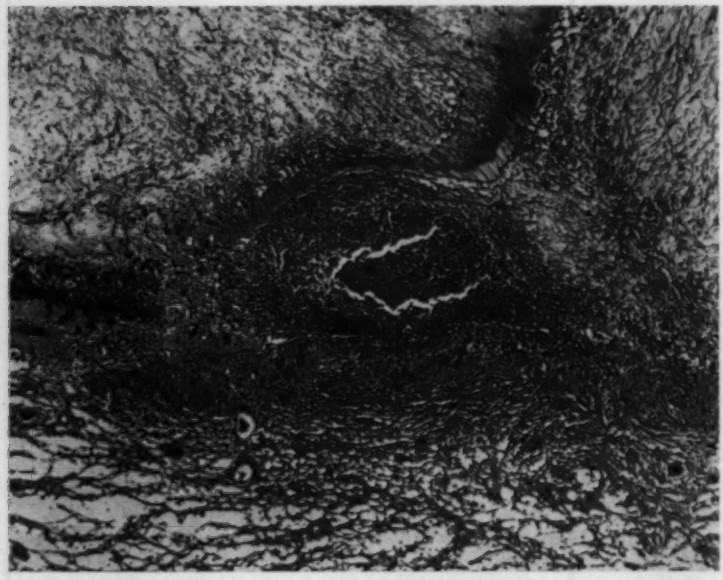

Fig. 14 (Woods and Wahlen). Granulomatous lesion of peripheral fundus. Etiology unknown. (A.F.I.P. #883824.)

six months after they had been injected into the eye.

Judging by this, one would not expect to find the fungus spores in the atrophic lesions of human eyes, but one would expect to find them in the fresh lesions of the progressive, fatal human disease. However, fatal histoplasmosis is extremely rare and in only one case have acute ocular lesions been reported. While this case came to autopsy, the eyes were not obtained and this golden opportunity was lost. One must therefore probably await the advent of a similar case before Koch's fundamental postulate can be fulfilled.

C. The small, discrete, peripheral scars noted in these eyes with a presumed histoplasmic uveitis have every appearance of being the residua of small granulomatous lesions. Small, discrete, peripheral granulomas have been found[17] in eyes enucleated for other reasons, but their etiology has never been demonstrated. Figure 14 illustrates such a lesion. It is reasonable to suppose that if the uveitis here reported is of a histoplasmic etiology, the peripheral scars are the residua of similar small granulomas caused by a tissue invasion by the H. capsulatum during the initial period of parasitemia.

D. The central, cystic lesions are characterized by edema, a serous effusion, and often by a later hemorrhagic extravasation. They appear quite different from the discrete, peripheral lesions. While they almost certainly have the same etiology in that they are related to the same basic infection, they may well have a quite different pathogenesis.

In the 1958 May Lecture, Maumenee[18] discussed at length the pathogenesis of disciform macular lesions. He pointed out that the peculiar vascular arrangement at the fovea might well predispose the eye to central cystic lesions. However, he emphasized that many of these disciform lesions, especially those in the older age group, were undoubtedly degenerative. In these patients ophthalmoscopic examination revealed no

evidence of a previous fundus inflammation, and histologic examination usually showed a break in Bruch's membrane, sclerotic changes in the choroidal vessels, subretinal hemorrhage, and little or no evidence of an inflammatory reaction. In other patients with disciform disturbances at the macula, especially those in the younger age group, ophthalmoscopic examination did show definite evidence of former inflammatory fundus disease, and in many of these patients the medical survey disclosed evidence of various systemic granulomatous diseases. Maumenee then emphasized that while the serous-hemorrhagic-glial response in these cases might well be a postinflammatory phenomenon, that this response was not limited to any single phlogogenic agent. He suggested that choroidal inflammation *per se* might affect the vascular supply of the fovea in some manner which would precipitate a serous or hemorrhagic reaction.

With these facts and premises in mind, the following hypothesis for the pathogenesis of this presumed histoplasmic uveitis may be advanced.

The uveal tract becomes infected during the generalized parasitemia which is believed to characterize the initial phase of benign histoplasmosis. The ocular reaction to this infection is the formation of small granulomas in the peripheral choroid or pars plana. These lesions gradually heal and become atrophic. They cause no symptoms and the patient is unaware of them. The invading spores either die, are killed off, or a few may survive and become encapsulated. These small granulomas are the cause of the small, discrete, peripheral scars. The later lesions at or near the fovea may be explained in one of two ways, either as late, atypical granulomatous lesions or as hypersensitive reactions.

On the first hypothesis, with any later depression in the patient's immunity, any surviving parsites would again propagate and multiply. If, in some unexplained manner, these multiplying spores became localized in the perifoveal region, they would cause a fresh lesion, the essential granulomatous nature of which would be masked by the localized serous effusion to which the macula is predisposed. Any later hemorrhage would be the result of necrosis involving the capillaries.

The second explanation—that the later central lesions are hypersensitive phenomena—is by far the more probable. The H. capsulatum is notoriously a great tissue sensitizer, and a prior infection of the uveal tract would undoubtedly leave behind a highly sensitized tissue. A late exacerbation of one of the peripheral fundus lesions which freed a few spores in the uvea, or a similar exacerbation of a remote systemic lesion would release antigen and result in a hypersensitive reaction in the sensitized uvea. This, like other hypersensitive reactions, would be characterized by a nongranulomatous type of inflammation of which a serous effusion is one of the characteristics. If this reaction occurs in the fundus, on account of the peculiar anatomic vascular arrangement at the fovea, it would be expressed as a localized serous effusion or as a central serous retinopathy. If the reaction were intense, or if there were an Arthus type of sensitivity, it might be complicated by hemorrhage. If the original granulomatous lesion were in the pars plana or in the iris, a later hypersensitive reaction would be manifested as an acute iridocyclitis. In either case, the hypersensitive reaction would persist until either the antigenic supply or the tissue hypersensitivity was exhausted. It is this late hypersensitive reaction which brings the patient to the ophthalmologist when the only clues to its etiology are the histoplasmic sensitivity and the tell-tale peripheral scars.

Admittedly, this explanation of the late lesions is hypothetical, but nevertheless it is logical and in accord with the clinical observations. It is strongly supported by the focal reaction around the central lesion after a diagnostic injection of histoplasmin. This was observed by Krause and later by us.

However this may be, when all hypothetical reasoning is abandoned and the various objections given due considerations, there remains a hard core of clinical observation and circumstantial evidence which permits the following conclusions:

Conclusions

1. There is an increased incidence of positive histoplasmin reactors in patients with granulomatous uveitis when compared to those with nongranulomatous uveitis and to the normal population.

2. Nineteen of the 62 patients with granulomatous uveitis here reported showed a peculiar and consistent pattern of ocular lesions. This consisted of discrete, atrophic, sparsely pigmented or unpigmented, peripheral lesions followed by a later cystic lesion at or adjacent to the macula. Approximately one half of these central lesions were complicated by a secondary surrounding hemorrhagic reaction.

3. Nine of these patients showed the trilogy of histoplasmin sensitivity, pulmonary calcification and anergy to tuberculin. In these nine patients the medical survey had revealed no cause other than histoplasmosis to which the uveitis might be attributed. In the other 10 patients the picture was not so clear-cut, but the ocular picture was characteristic and the patients had undoubtedly had a benign systemic histoplasmosis.

4. While it is, of course, possible that this fundus picture may be caused by some other unsuspected etiologic agent, it has thus far been observed only in patients who are hypersensitive to histoplasmin.

5. Based on this evidence, it appears reasonable to assume a cause-and-effect relationship between a benign systemic histoplasmosis and these ocular lesions. There is also some evidence that a similar relationship may exist between histoplasmosis and an occasional case of nongranulomatous iridocyclitis.

The Johns Hopkins Hospital (5).

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LONG-TERM RESULTS IN GLAUCOMA THERAPY*

THE SANFORD R. GIFFORD LECTURE

PAUL A. CHANDLER, M.D.
Boston, Massachusetts

Few ophthalmologists have had the privilege of having a lecture named in their honor. Such an honor is not bestowed lightly. The individual so honored must be one who has made notable contributions to ophthalmology and, furthermore, he must have a character that evokes the affection and respect of his colleagues. Such a man was Dr. Sanford R. Gifford.

I consider it a great honor to have the privilege of presenting to you this Gifford lecture.

It is now an almost universally accepted fact that there are two types of glaucoma—open-angle glaucoma and angle-closure glaucoma. The elevated tension in both types is due to obstruction to outflow from the anterior chamber but the mechanism of the obstruction is quite different in the two types. In open-angle glaucoma aqueous has access to the filtration apparatus at all times. The obstruction to outflow resides in the filtration apparatus itself. In angle-closure glaucoma the filtration apparatus is normal. Obstruction to outflow is due solely to closure of the angle.

This concept has led to marked improvement in recent years in the treatment of angle-closure glaucoma. It is generally recognized that the treatment of this type of glaucoma is primarily surgical, and early surgical intervention has provided most gratifying results. Long-term results in the therapy of angle-closure glaucoma are generally excellent if proper treatment is employed. Serious loss of vision in this form of glaucoma is usually the result of neglect on the part of the patient, improper diagnosis leading to improper treatment, and delay in surgical intervention.

No similar great advance has taken place in recent years in the treatment of open-angle glaucoma. Though a few new miotics have been added to our medical armamentarium, their effectiveness is generally not strikingly better than that of the older drugs. Epinephrine bitartrate as an adjunct to miotic therapy has proven very helpful in many cases. The discovery of the carbonic anhydrase inhibiting drugs as a therapeutic agent in glaucoma is perhaps the most important advance in recent years in the treatment of open-angle glaucoma. With the addition of these agents, operation can in many cases be deferred or avoided altogether. The judicious application of these various medical agents in a given case is of utmost importance. Failure of medical treatment may often be due to inadequate use of these agents. Nevertheless, there remain cases of open-angle glaucoma in which the maximal medical treatment fails to maintain tension in a range sufficiently low to prevent progressive loss of function of the eye, and surgical treatment must be advised.

How long may medical treatment maintain the function of the eye, and if surgical treatment is undertaken, how long may it be effective in controlling the glaucoma?

It is the purpose of this communication to review the results of treatment over a long period of years in the hope that these observations may be helpful in planning long-term therapy. It was felt that a statistical analysis of a large number of cases would be of less value than a detailed study of a lesser number of individual cases. If, in a statistical analysis of cases, one reports that in a certain group treatment was successful, and in another group unsuccessful, the question of the cause of the success or failure may remain unanswered; whereas, in a study of individual cases the cause for success or

* From the Massachusetts Eye and Ear Infirmary.

failure may be more readily discernible. The 20 cases in this study were selected to illustrate the results of medical and surgical treatment applied at various stages of glaucoma in patients who have been observed over a long period of years. They may be divided into four groups:

I. Cases 1 through 10. In this group there was successful surgical treatment of one or both eyes.

II. Cases 11 through 14 and the right eye in Case 7. In this group only medical treatment was employed.

III. Cases 15 through 17. In this group operation was employed in one or both eyes but the operation was unsuccessful or only partly successful, and medical treatment was continued.

IV. Cases 18 through 20. This group consists of cases of low-tension glaucoma or pseudoglaucoma that were successfully treated by operation.

It can be noted that cases with completely unsuccessful filtering operations where the tension remained high are not included, since it goes without saying that in such cases the surgeon will try repeated surgical intervention until a successful result is obtained or the eye has lost all vision. Probably most of the cases presented are of open-angle glaucoma but, since gonioscopy was first done many years after surgery, it is difficult to be sure in all the surgically treated cases. All the cases treated medically were of open-angle glaucoma.

Group I

Case 1

This patient, a woman, at 52 years of age was first examined by the late Dr. George S. Derby in 1931. There was a history of an operation for glau-

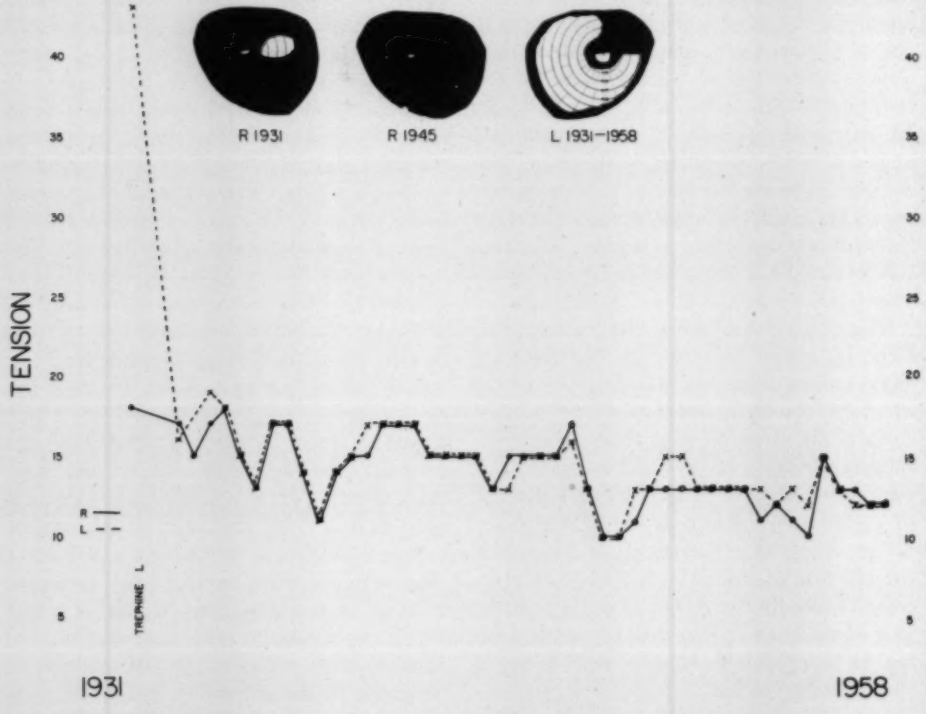

Fig. 1 (Chandler). Average postoperative tension: R.E., 14 mm. Hg; L.E., 14 mm. Hg. Loss of small peripheral field in the right eye, no loss of field in the left eye. Period of observation, 27 years.

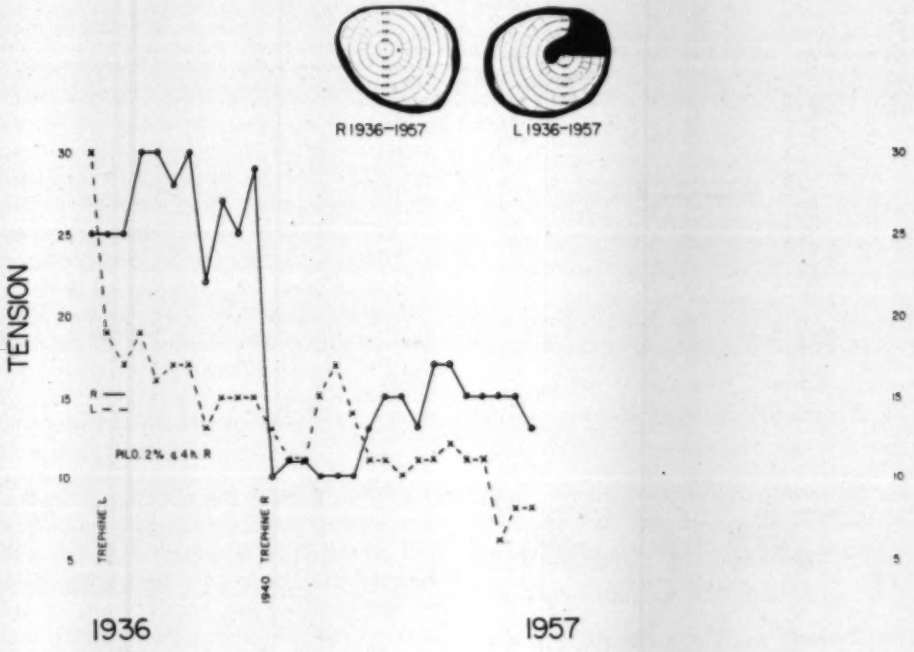

Fig. 2 (Chandler). Average postoperative tension: R.E., 13.2 mm. Hg; L.E., 13 mm. Hg. No loss of field either eye. Period of postoperative observation: R.E., 17 years; L.E., 21 years.

coma on the right eye seven years previously. Corrected vision was: R.E., 6/15; L.E., 6/5. In the right eye there was marked bulging of the conjunctiva above the limbus, with some pigment under it, suggesting a previous iridencleisis. Media were clear, the disc markedly cupped.

Field studies showed a small central field and a small temporal island remaining (fig. 1). In the left eye the disc was markedly cupped and there was considerable loss of field (fig. 1). Tension was R.E. 19 mm. Hg.*

A trephining operation was done on the left eye by Dr. Derby. A good filtering scar developed and, as seen in the tension curve, tension in both eyes has been maintained in the low normal range. The temporal island of vision in the right eye disappeared 20 years later. The small central island remained as long as it could be accurately measured. Increasing nuclear lens opacity has precluded ordinary perimetry in this eye for several years but there is still a central island of vision to hand movements. The field in the left eye has remained almost exactly the same. The field taken in 1958 is almost a carbon copy of the initial field taken in 1931. Twenty years after operation, in spite of mild nuclear sclerosis, corrected vision was 6/6— in the left eye. The nuclear sclerosis has gradually increased and there are now some senile macular changes. In 1958, corrected vision in the left eye was 6/15. The patient is now 79 years of age.

Comment. This case illustrates almost complete control of glaucoma by a filtering operation for 34 years in the right eye, 27 years in the left. The tension curve (fig. 1) demonstrates maintenance of a low normal tension postoperatively, with a tendency for the tension to become gradually lower over a period of years. The fact that the field in the left eye has remained almost exactly the same for 27 years demonstrates that in some cases of moderately advanced glaucoma with marked cupping of the disc further loss can be prevented if the tension is maintained at a sufficiently low level. It can be noted that

* All tension records are Schiøtz. Up to 1929 the old Schiøtz tonometer was used. This tonometer with the scale used at that time gave a reading two or three mm. Hg lower than the new Schiøtz tonometer, which with a revised scale was used after 1929. The 1948 scale has been used since it became available.

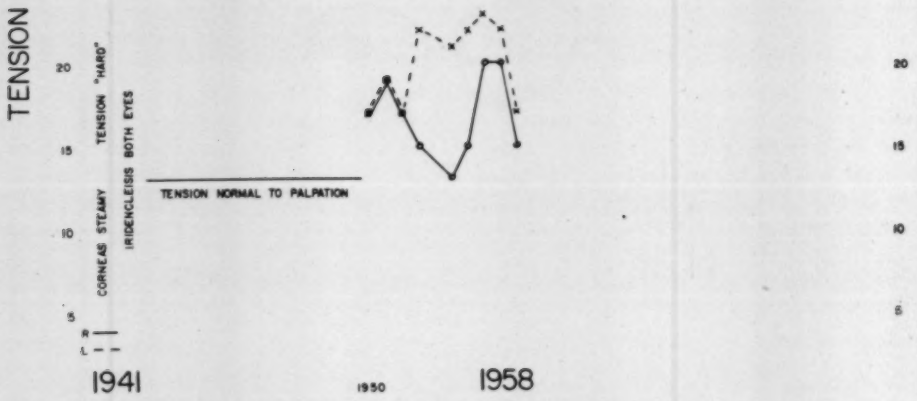

Fig. 3 (Chandler). Congenital glaucoma. Operation at the age of five months. No damage from glaucoma. Period of postoperative observation, 17 years.

for the most part the postoperative tension in this case has been 15 mm. Hg or less with a tension average of 14 mm. Hg in each eye.

CASE 2

This woman was first examined in 1936 at the age of 48 years. She has been under treatment for glaucoma for two weeks. Corrected vision was 6/5 in each eye. Tension was: R.E., 25 mm. Hg; L.E., 30 mm. Hg. The right disc was normal, the field full. In the left eye the disc was cupped, especially below and temporally. The nasal border of the disc was normal.

Field studies revealed loss of most of the upper nasal field and an upper Bjerrum scotoma (fig. 2). A trephining operation was done on the left eye. A good filtering scar developed, and as seen in the tension curve, tension has been consistently low postoperatively. The last field, taken in 1957, was exactly the same as the original field.

The right eye was treated with two-percent Pilocarpine. During the three years between 1937 and 1940, tension in this eye generally ranged from 25 to 30 mm. Hg. By 1940 the right disc showed early cupping but there was no field defect. A trephining operation was done on the right eye in 1940. As can be seen in the tension curve, tension has been consistently low, though generally slightly higher than in the left eye. No further change has taken place in the appearance of the disc, and the field remains full. In 1957, when the patient was last examined, corrected vision in the right eye was 6/6, in the left 6/12. There was some nuclear sclerosis in both eyes.

Comment. This case is an example of complete arrest of the glaucoma for a period of 20 years in the right eye, 17 years in the left, as a result of successful filtering operations. It is striking that there has been no loss of field whatever in 20 years in the already damaged left eye, and the field in the right eye has remained full for 17 years. It can be noted that the average postoperative tension was 13.2 mm. Hg in the right eye, 13 mm. Hg in the left. The left eye in this patient demonstrates the fact that if tension is maintained at a sufficiently low level, there need be no further loss even in a damaged eye. In the light of modern methods of therapy, one can feel that more vigorous medical treatment might have been employed in the right eye before resorting to operation.

CASE 3

This male patient at five months of age was first examined in 1941. Corneas were 12.5 mm. in diameter, steamy throughout. Tension was not measured with a tonometer, but was "hard" to palpation. An iridencleisis was done on both eyes. A considerably elevated bleb developed in both eyes. During the next nine years the tension was not measured with a tonometer but was always normal to palpation. Beginning in 1950 the tension was measured with a tonometer, and the results are pictured in the tension chart (fig. 3).

The left cornea cleared entirely after operation, but a bandlike opacity remains in the right eye, the site of an old ruptured Descemet's membrane. Both discs are normal in color and there is no pathologic cupping. There is mixed astigmatism in both eyes, 4.5 diopters in the right, 8.0 diopters in the left. Corrected vision is 6/9 in the right eye, 6/15 in the

left. This patient has been active in sports and has an excellent scholastic record.

Comment. Filtering operations for congential glaucoma have now been largely supplanted by goniotomy and goniopuncture. The results of filtering operations have been much less satisfactory in congenital glaucoma than in the adult form. Nevertheless, many ophthalmic surgeons can attest that filtering operations may be completely successful in arresting the disease. This case is included as an example of such a successful result. Filtering operations for congenital glaucoma may still offer a chance of success in cases in which repeated goniotomy and goniopuncture have failed, and therefore should not be entirely discarded.

CASE 4

This man at age of 65 years was first examined in 1939. Corrected vision in the right eye was 6/5. The left eye was totally blind. Tension was: R.E., 22 mm. Hg; L.E., 50 mm. Hg. The right disc showed cupping at the lower outer pole. The left disc was deeply and totally cupped. Field studies in the right eye showed an upper Bjerrum scotoma and loss of most of the upper nasal field (fig. 4). A tension study was carried out. It was found that without treatment tension ranged from 22 to 32 mm. Hg, with the highest readings in the middle of the day. Under two-percent Pilocarpine every four hours tension did not go above 21 mm. Hg. The patient was continued on two-percent Pilocarpine. Until 1942 tension remained 25 mm. Hg or less at all examinations. In January, 1942, a tension of 41 mm. Hg was found. Pilocarpine was increased to four percent. As can be seen from the tension curve (fig. 4), tension was out of control and an iridencleinsis was done.

A good filtering scar developed which has con-

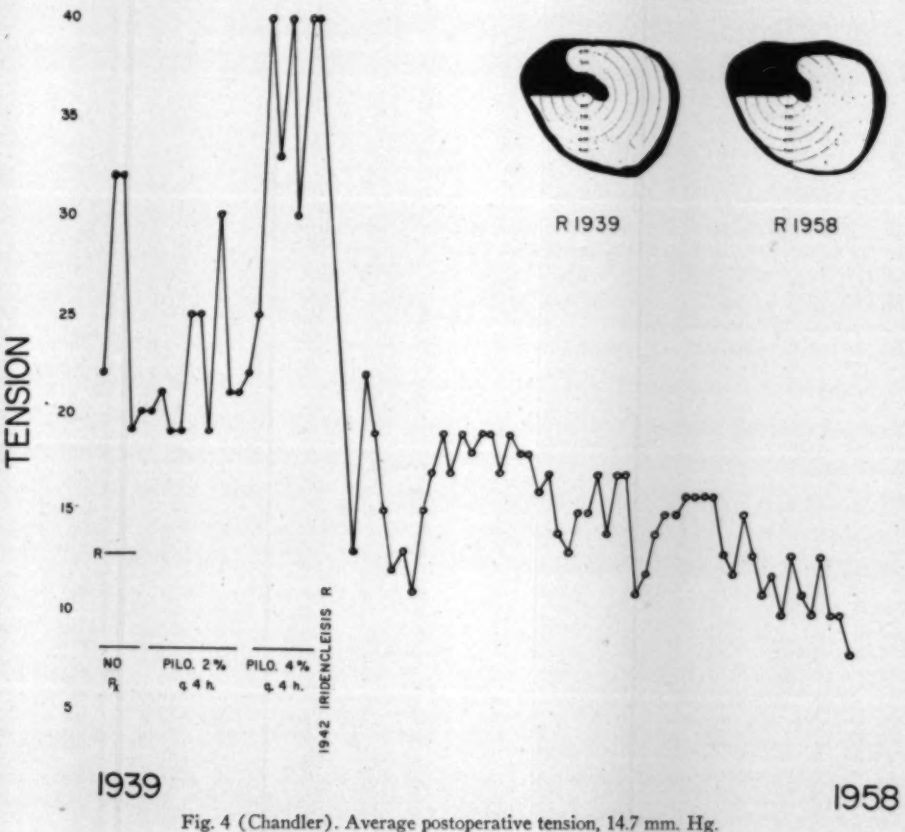

Fig. 4 (Chandler). Average postoperative tension, 14.7 mm. Hg. Period of postoperative observation, 16 years.

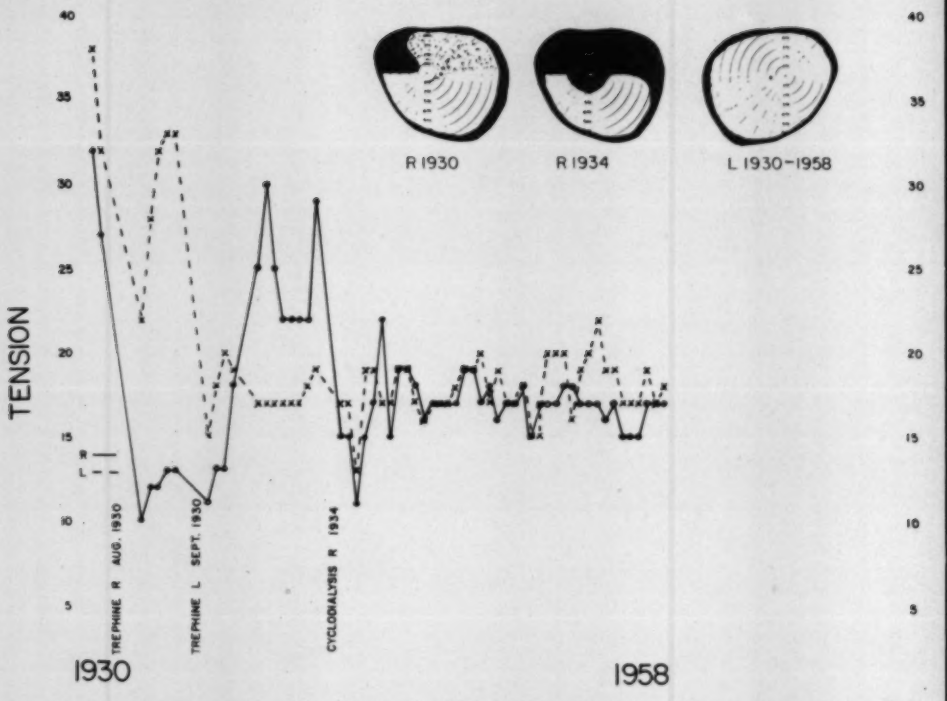

Fig. 5 (Chandler). Average postoperative tension: R.E., 17 mm. Hg; L.E., 17.7 mm. Hg. No loss of field in the left eye. Considerable loss in right eye prior to last operation. Period of observation, 28 years.

tinued to function well to the present time. Postoperatively tension has been generally low and tending to become lower of late years. The average postoperative tension was 14.7 mm. Hg. In 1958, there was fairly dense nuclear sclerosis. Corrected vision was 6/15. As can be seen in the field charts, there has been minimal loss of field during the 16 years following operation. The patient is now 84 years of age.

Comment. This case illustrates again the fact that a successful filtering operation may preserve the field of an already damaged eye over a long period of years.

CASE 5

This woman was first examined at the age of 43 years by the late Dr. George S. Derby, in 1922, for refraction. She was seen at intervals until August, 1930, when the first abnormality was noted. Corrected vision in the right eye was 6/6, in the left 6/5. There was cupping of the lower half of the right disc, with atrophy. The left disc was normal. Tension was: R.E., 35 mm. Hg; L.E., 45 mm. Hg. The left field was full. In the right eye there was loss of most of the upper nasal field and the remaining upper field was dim (fig. 5). Two-percent Pilocarpine was prescribed. A trephination was done on the right eye by Dr. Derby and one month later on the left eye. A filtering scar developed in both eyes and the tension became soft. Since the patient's home was in Illinois, she was followed part of the time by her local ophthalmologist and therefore was not seen frequently in our office, usually once a year.

In July, 1933, tension in the right eye was found to be 25 mm. Hg, and there was less evidence of filtration. By February, 1934, there was loss of the remaining upper field, and a lower Bjerrum scotoma had developed, isolating a tiny central field. Vision was 6/60. A cyclodialysis was done on the right eye by the late Dr. Harry Gradle. Thereafter tension has been well controlled in this eye (fig. 5).

Within a year following the cyclodialysis operation the lens became opaque. Therefore the last field recorded was in 1934. In the left eye a good filtering scar remains and tension has been well controlled since the operation in 1930. In 1958, corrected vision in the left eye was 6/9. There was considerable nuclear sclerosis. There was no pathologic cupping, and the field remained full. The patient is now 79 years of age.

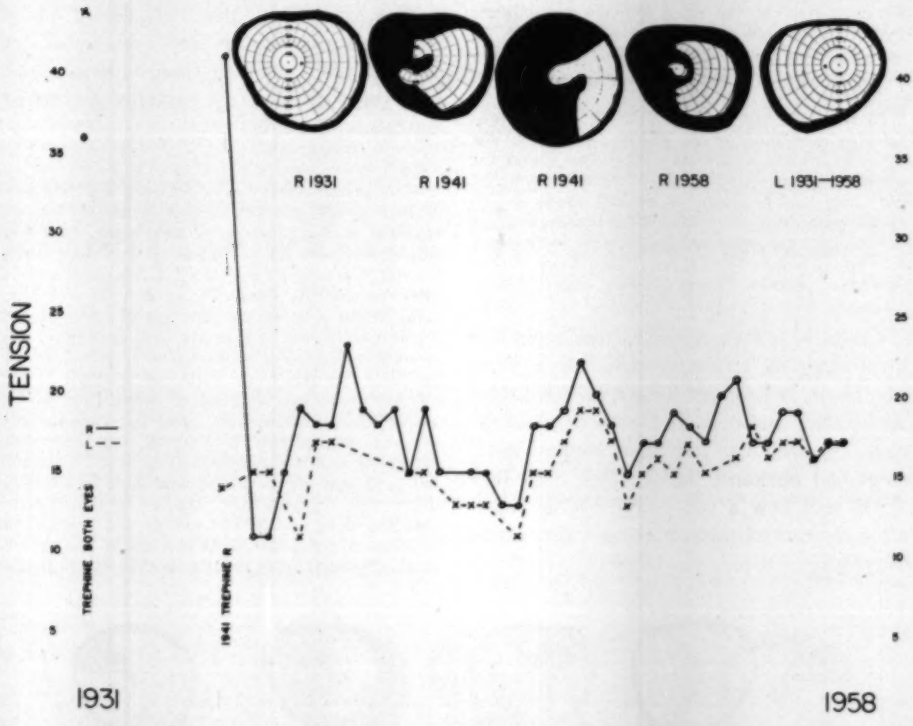

Fig. 6 (Chandler). Average postoperative tension: R.E., 17 mm. Hg; L.E., 15.4 mm. Hg. Slight loss of field in right eye after second operation. No loss of field in the left eye. Period of observation, 27 years.

Comment. The trephination in the right eye failed to function except for a short time and there was considerable field loss between 1930 and 1934, demonstrating that an eye with a cupped disc withstands increased tension poorly. One might argue that in this case medical treatment might have been given a longer trial in the left eye before resorting to operation, since the disc was normal. It could well be that this eye might have tolerated a tension of 30 to 35 mm. Hg over a period of years. However that may be, this patient has preserved a full field for 28 years, there is no more lens opacity than is often seen at this age, and it must be concluded that operation was the wisest choice for long-term therapy in this case. There is no way of judging the status of the right eye except by the tension.

CASE 6

This female patient at 15 years of age was first seen by the late Dr. George S. Derby in 1931. Her mother had glaucoma and had had operation at the age of 15 years. The maternal grandfather and great grandfather had glaucoma.

When this patient was first examined, vision was 6/5 in each eye. Anterior chambers were of normal depth, fundi were normal, fields full, tension was: R.E., 36 mm. Hg; L.E., 41 mm. Hg. A trephination was done on both eyes by Dr. Derby. One month after operation there was a filtering scar in both eyes and the tension was soft.

The patient then went under the care of another ophthalmologist and was next seen by me in 1941. Some time during this interval the trephine opening in the right eye had closed. There was cupping of the disc and a considerable field defect in this eye (fig. 6). Tension was 41 mm. Hg. In the left eye there was a good filtering scar; tension was 14. mm. Hg. The disc was normal, the field full. Corrected vision was: R.E., 6/6+; L.E., 6/5.

Another trephining operation was done on the right eye. A filtering scar developed. As can be seen in the tension curve (fig. 6) tension has

generally been in the normal range but slightly higher in the right than in the left. In 1958, 27 years after operation on the left eye, 17 years after the second operation on the right eye, conditions are as follows:

In the right eye, there is a small localized posterior cortical lens opacity. The disc is cupped. The field (fig. 6) has shown very little change over the years. Corrected vision is 6/6. In the left eye, media are clear, fundus normal, corrected vision is 6/5. The occasional peaks of somewhat elevated tension in the right eye were associated with spontaneous anterior chamber hemorrhage which came during menstrual periods. These episodes ceased several years ago.

Comment. This is a case of true hereditary glaucoma. Due to the youth of the patient, Dr. Derby wisely chose surgical treatment at the start, and in 1941 I reoperated on the right eye without delay since medical treatment had obviously failed. This case illustrates well how a successful filtering operation can control glaucoma over a long period of years.

This patient has three sons, one of whom developed glaucoma in both eyes at the age of eight years. In both eyes the tension has been controlled for two years by goniotomy and goniopuncture.

CASE 7

This woman at the age of 36 years was first seen in 1910 by the late Dr. George S. Derby, for refraction. Nothing abnormal was noted. She was seen periodically for refraction until in June, 1926, the first abnormality was noted. Corrected vision in each eye was 6/6. Tension was: R.E., 25 mm. Hg; L.E., 35 mm. Hg. In the right eye the disc showed a physiologic cup, but in the left eye there was cupping and atrophy at the lower outer margin of the disc. The field in the right eye was full. In the left eye there was slight nasal constriction in the peripheral field and a large upper Bjerrum scotoma (fig. 7). One-percent Pilocarpine was prescribed. As can be seen in the tension curve (fig. 7), tension fell to 25 mm. Hg in the left eye, 22 to 24 mm. Hg in the right. In September, 1926, a trephining operation was done on the left eye by Dr. Derby. An excellent filtering scar developed, and as can be seen in the tension curve, the tension in the left eye

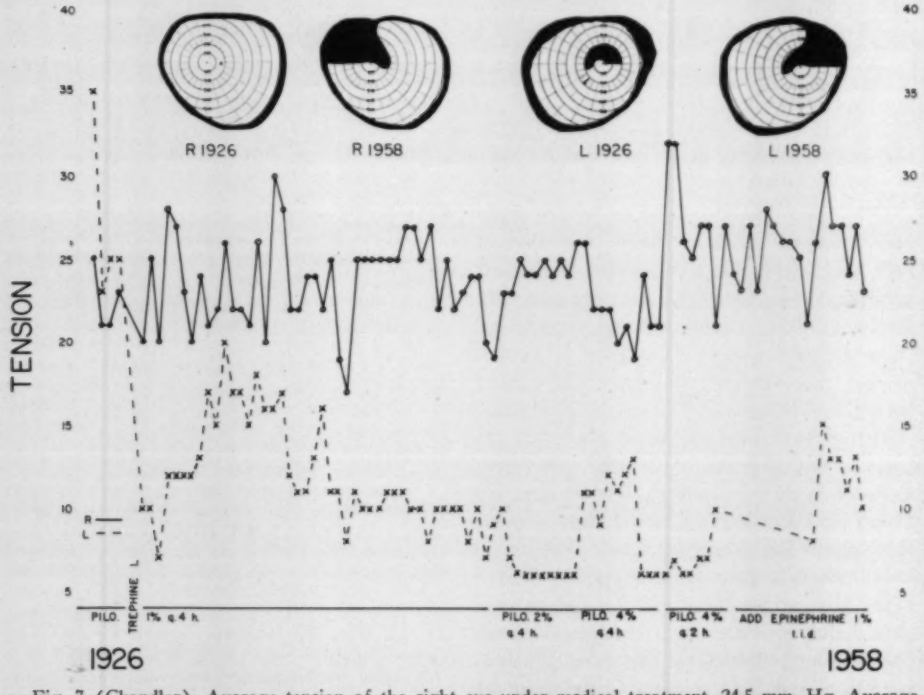

Fig. 7 (Chandler). Average tension of the right eye under medical treatment, 24.5 mm. Hg. Average postoperative tension of left eye, 10.4 mm. Hg. Loss of one quadrant of field in right eye, slight loss in left eye. Period of observation, 32 years.

during the next 32 years was always low. Pilocarpine (one percent, every four hours) was continued in the right eye. In 1946, this was increased to two percent, in 1949, to four percent. Beginning in 1955, four-percent Pilocarpine was used every two hours. In 1956 Epinephrine bitartrate (1.8 percent, t.i.d.) was added.

As can be seen from the tension curve, tension has averaged about 25 mm. Hg with some tendency to become higher in the last three years. The field remained full in the right eye until 1946 when for the first time a small upper Bjerrum scotoma was noted. Since that time there has been a gradual loss of field in this eye until in 1958, there is loss of most of the upper nasal field. By 1930, the Bjerrum scotoma in the left eye had broken through into the periphery and the field at that time was almost exactly the same as the field taken 28 years later in 1958 (fig. 7). In 1958, there was nuclear sclerosis in both eyes. Corrected vision in the right eye was 6/15+; in the left 1/60, 6/30 with pinhole. There was still an excellent filtering scar in the left eye. The patient is now 84 years of age.

Comment. This case illustrates almost complete control of glaucoma over a period of 32 years in an eye in which tension was maintained at quite low levels, following a successful filtering operation. The behavior of the right eye illustrates the fact that if the disc is normal at the beginning of treatment, damage can be prevented over a period of many years even with a tension in the mid twenties. It seems altogether probable that the left eye in this patient would not have tolerated the level of tension maintained in the right eye without progressive loss of field. Field loss in the right eye may progress but it is probable that medical treatment will preserve a useful field in this eye during the rest of the patient's life.

CASE 8

This man at 50 years of age was first examined by the late Dr. George S. Derby in 1927. Corrected vision was 6/9 in the right eye, 6/12 in the left. The right disc was totally cupped and there was marked field loss (fig. 8). In the left eye there was no cupping of the disc, but the disc outlines were blurred and there were many retinal hemorrhages

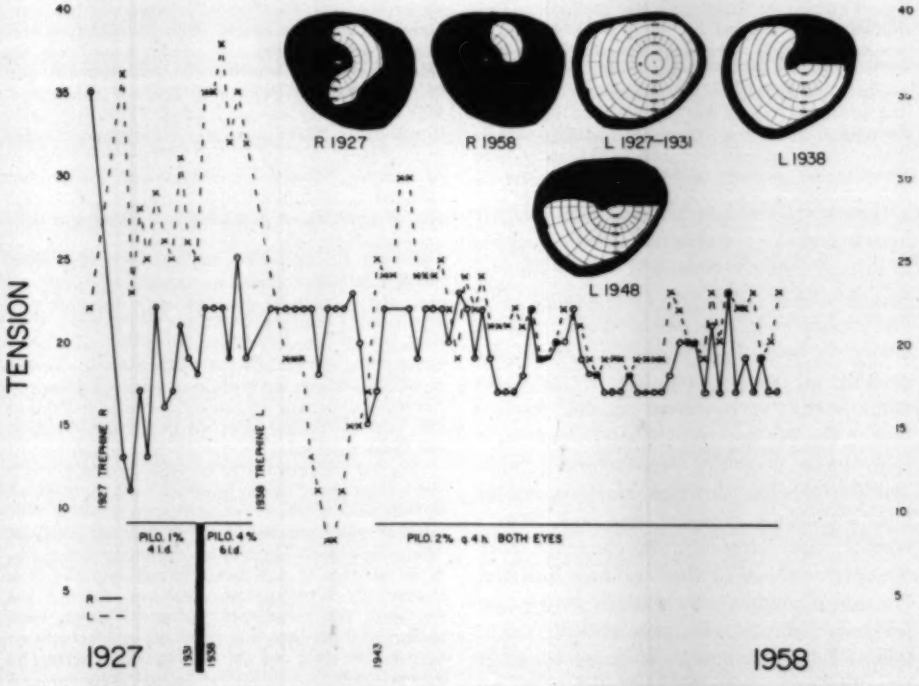

Fig. 8 (Chandler). Average postoperative tension: R.E., 19 mm. Hg; L.E., 20.2 mm. Hg. Loss of half of peripheral field in right eye in 31 years with preservation of the tiny central island. Loss of one quadrant of the field in the left eye in 10 years. Total period of observation, 31 years.

suggesting partial occlusion of the central vein. The field was full. Tension was: R.E., 35 mm. Hg; L.E., 22 mm. Hg. A trephining operation was done on the right eye by Dr. Derby. A filtering scar developed which has continued to function to the present time, with tension rarely higher than 22 mm. Hg. (fig. 8). The tiny central field has remained practically unchanged for 31 years though there has been some loss in the peripheral field. In 1958 corrected vision was 6/9.

Following operation on the right eye, tension shortly rose to 36 mm. Hg in the left eye. One-percent Pilocarpine every four hours was prescribed for the left eye, and this maintained the tension during the next four years for the most part under 30 mm. Hg. In 1931, the field in the left eye was full.

The patient was not seen again until 1938, seven years later. This gap is indicated on the chart by the heavy black vertical line. During these seven years the patient had been under the care of another ophthalmologist and had continued to use a miotic. When seen again in 1938, tension in the left eye was 35 mm. Hg. There was loss of the upper nasal field (fig. 8). Four-percent Pilocarpine every three hours was prescribed, but the tension remained over 30 mm. Hg and a trephining operation was done. During the next four years, tension in the left eye was well controlled, but in 1943 it rose to 30 mm. Hg, due to partial closure of the trephination bleb. Two-percent Pilocarpine was prescribed. Tension gradually fell and for the past 10 or 12 years has been at fairly satisfactory levels, though generally somewhat higher than in the right eye. Vision gradually failed in the left eye due to increasing lens opacities and, by 1949, vision was reduced to counting fingers at a few feet. The last field in 1948 showed some further loss in the upper field. There is still evidence of filtration in both eyes.

Comment. The loss of field in the left eye over a period of seven years points out the failure of medical treatment. Since the disc was normal in 1931, one must suppose that the tension had been well over 30 mm. Hg during the years 1931 to 1938 to have caused cupping of the disc and loss of the upper nasal field. The continued gradual loss of field in the left eye over the next 10 years, in spite of a generally satisfactory tension, demonstrates the fact that usually a cupped disc withstands pressure less well than a normal disc. However, the average postoperative tension in this eye was 20.2 mm. Hg, considerably higher than in some of the previous patients who showed little or no loss of field after operation. A tension of 20 mm. Hg will generally be found to be too high for a considerably damaged eye. The result in the right eye is striking. It is remarkable that the small central island has been preserved for 31 years.

CASE 9

This woman at the age of 46 years was first examined in 1942. The left eye was totally blind after two unsuccessful operations for glaucoma. The anterior chamber in this blind eye was flat. There was some suggestion of filtration above and tension was 30 mm. Hg. In the right eye corrected vision was 6/5. Tension was 49 mm. Hg. The anterior chamber was moderately shallow. The disc showed considerable cupping and atrophy and field studies showed considerable loss of field (fig. 9).

Since the patient was then on miotic therapy, operation was advised. A trephining operation was done on May 15, 1942. Vitreous was lost through the trephine opening. The following day there was no evidence of filtration, the anterior chamber was extremely shallow, and there appeared to be uveal tissue in the trephine opening. On May 18th tension was firm. The flap was again turned down, the uveal tissue in the trephine opening was pulled out and excised. This was followed by further vitreous loss. The anterior chamber became formed on the fifth postoperative day.

During the next three months there was very little evidence of filtration and tension under four-percent Pilocarpine ranged from 40 to 17 mm. Hg, usually under 30 mm. Hg. On August 26th the anterior chamber was very shallow, tension was 45 mm. Hg. On September 2nd, an iridencleisis with sclerectomy was performed. There was again vitreous loss. Three days later the anterior chamber was formed and tension soft. There was evidence of filtration. However, one week later the anterior chamber was again extremely shallow and tension was 35 mm. Hg. A posterior sclerotomy was done on September 12th.

During the next four months there were many anxious moments. Tension ranged from 17 to 40 mm. Hg in spite of massage and treatment with four-percent Pilocarpine. At last by February, 1943, tension had fallen to 22 mm. Hg and was never again found elevated (fig. 9). The anterior chamber remained shallow, but there was definite evidence of filtration at the site of the iridencleisis, and this is still evident. Nuclear sclerosis gradually developed, and vision declined.

The last recorded field in 1950 showed a slight loss as compared to the original field. In 1955, an intracapsular cataract extraction was done. The vitreous was completely fluid and the eye collapsed after the section. The section was made to the side, in an attempt to avoid interfering with the bleb. Fortunately filtration continued and the eye has remained soft. Corrected vision is 6/6—. Field studies have not been done since the cataract extraction, but at least one can be sure that the central field is intact.

Comment. In retrospect it seems probable

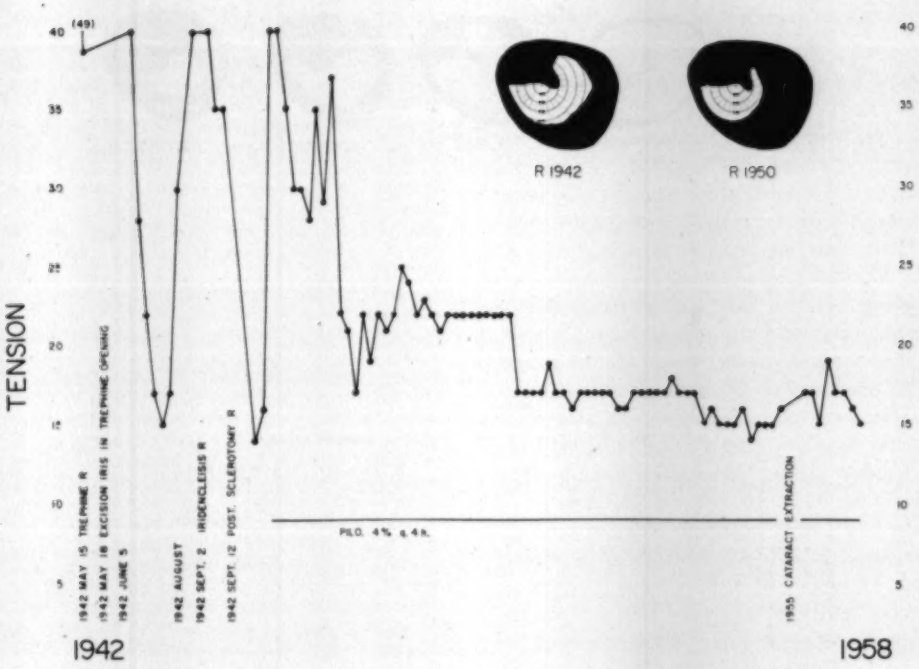

Fig. 9 (Chandler). Average postoperative tension after the first operation, 18.3 mm. Hg. Somewhat lower average after last operation. Slight loss of field. Period of observation, 16 years.

that this was a case of malignant glaucoma postoperatively. Lens extraction after the second operation might have been the wiser procedure. It is noteworthy that there was vitreous loss at every operation on each eye. By great good fortune the iridencleisis finally functioned well, and has kept the tension low. Perhaps the principal lesson in this case is "don't give up the ship," though it would appear that there may have been more good luck than science involved in arresting this process.

CASE 10

This woman was first seen by the late Dr. George S. Derby in 1927. The patient was then 51 years of age. Examination at that time revealed a corrected vision of: R.E., 6/5; L.E., 6/12. The right disc showed no pathologic cupping, the field was full, tension 27 mm. Hg. In the left eye there was cupping of the disc with considerable loss of field (fig. 10). Tension was 31 mm. Hg. Pilocarpine was prescribed.

Though there is no note in the record, evidently the tension was not controlled in the left eye, for a trephining operation was done by Dr. Derby on this eye one month later. A filtering scar developed in this eye. As can be seen from the tension curve (fig. 10), tension in the right eye was not well controlled by medical treatment and, in 1928, a trephining operation was done on the right eye by Dr. Derby. A filtering scar developed in this eye also.

As can be seen in the tension chart, tension has been maintained for the most part under 20 mm. Hg for 30 years. The field in the right eye has remained full. In the left eye there has been a gradual loss of field over the years; see the initial field (fig. 10) and representative fields at 10-year intervals.

Nuclear sclerosis gradually developed in both eyes, earlier in the left, yet as late as 1947, 20 years after surgery, corrected vision was still 6/6— in the right eye, 6/30 in the left. In 1958 corrected vision in the right eye was 6/9—, 6/30 in the left.

Comment. One might argue that operation was done in the left eye without an adequate trial of medical treatment but the failure of medical treatment to control the tension in the right eye indicates that operation was the wiser choice at the outset. There is still

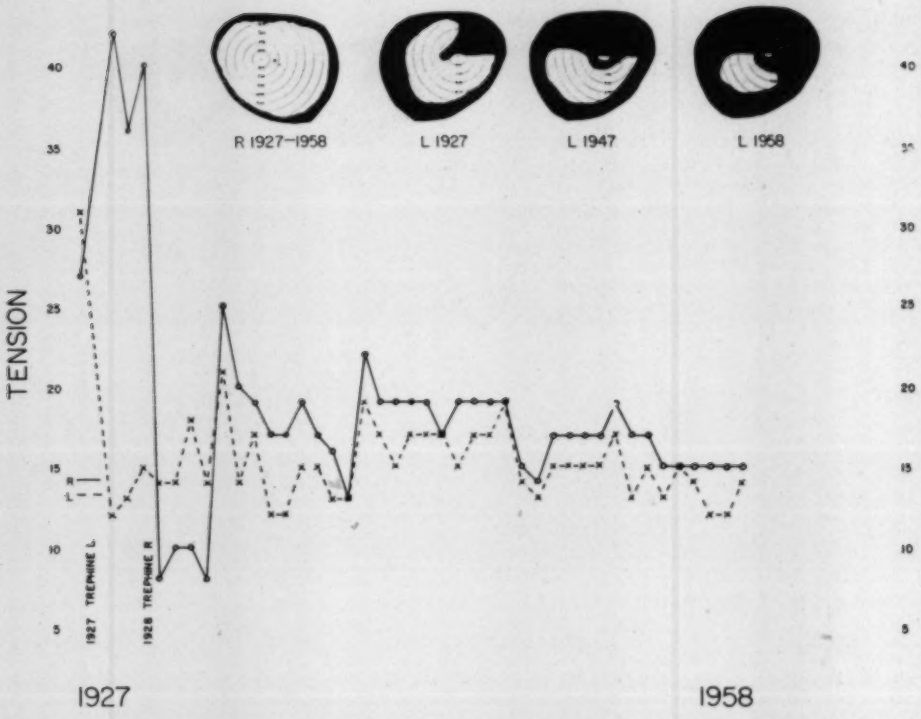

Fig. 10 (Chandler). Average postoperative tension: R.E., 16.6 mm. Hg; L.E., 15 mm. Hg. No loss of field in right eye, considerable loss in left eye. Period of observation, 31 years.

an obvious filtering scar in both eyes. It can be seen from the tension chart (fig. 10) that there has been adequate tension control, with an average postoperative tension of 16.6 mm. Hg in the right eye, 15 mm. Hg in the left. It is interesting to note that, although the average tension in the left eye has been generally slightly lower than in the right eye, there has been slow loss of field in the left eye, while the field in the right eye has remained full. This is evidence that a normal disc withstands pressure better than a cupped disc.

Group II

Case 11

This man at the age of 45 years was first examined by the late Dr. George S. Derby in 1922. At that time corrected vision was: R.E., 6/5; L.E., 6/6. Tension was R.E., 19, mm. Hg; L.E., 43 mm. Hg. The right disc was normal, the field full. In the left eye the disc was cupped and there was loss of most of the upper field. A trephining operation was done on the left eye by Dr. Derby. A good filtering scar developed and for the next four years tension in this eye ranged from 8.0 to 11 mm. Hg. In 1926 the filtering scar became infected, all treatment failed and the eye was enucleated.

From 1922 to 1936 tension in the right eye was controlled with one-percent Pilocarpine (fig. 11). During the following two years the tension became more difficult to control. Pilocarpine was increased to two percent and then to four percent. In 1938, a tension study was carried out with the patient receiving four-percent Pilocarpine every four hours. The readings were 40 mm. Hg at 8:00 A.M., 35 mm. Hg at noon, 26 mm. Hg to 30 mm. Hg at 6:00 P.M., 26 to 30 mm. Hg at 9:30 P.M. Epinephrine bitartrate (1.8 percent, three times a day) in addition to the miotic was prescribed, and another tension study carried out. The results were: 8:00 A.M. 30 mm. Hg, noon 28 mm. Hg, 6:00 P.M. 22 mm. Hg, 9:30 P.M. 17 mm. Hg. Three months later another tension study on the same medication revealed: 8:00 A.M. 22 mm. Hg, noon 26 mm. Hg, 5:00 P.M. 26 mm. Hg, 9:30 P.M. 19 mm. Hg.

The patient was continued on four-percent Pilocarpine every four hours and 1.8 percent epi-

nephrine bitartrate, t.i.d. The tension curve (fig. 11) illustrates the behavior of the tension over the years. On many occasions the peaks of elevated tension were found to be due to failure to use the drops on schedule. In 1954, when the tension was found to be 27 mm. Hg after having been in the low 20's for three or four years, 125 mg. of Diamox twice a day was prescribed. When Diamox was used regularly, tension was maintained in the low 20's until 1957 when a tension of 32 mm. Hg was found. A dosage of 250 mg. of Diamox twice a day was prescribed. Since then, tension has been maintained at 25 mm. Hg or less.

The peripheral field remained full to a three mm. object until 1947 when a small upper nasal step was noted. For two or three years prior to this there was some dimness of the upper field with a three-mm. target. In 1958, due to a one-mm. pupil and considerable nuclear sclerosis it was necessary to use a 10-mm. object to obtain a reproducible field. There appears to be a definite upper nasal step and some general constriction of the upper field (fig. 11). However, there is considerable lens opacity and some of the apparent defect may be due to the lens since the field is full by confrontation.

For some years due to the miosis and the lens opacity, it has not been possible to get a clear view of the disc. Central vision has gradually declined due to nuclear sclerosis. In 1946 vision was 6/9+, in 1950 6/12, in 1952 6/15, in 1956 6/15—, in 1958 6/18. Due to the extreme miosis the patient has never worn glasses. No lens improves the distance visión, and he still reads without glasses.

Comment. This case illustrates the favorable results of long-term medical treatment when the disc is normal and the field full at the outset. I have repeatedly observed that a normal disc appears to be more resistant to increased pressure than a deeply cupped disc. The beneficial effect of the addition of epinephrine bitartrate to miotic therapy is well illustrated in this case. In the tension studies the peak elevation of tension which occurred in this patient in the early morning was reduced from 40 to 30 mm. Hg and the tension measured later in the day was correspondingly reduced. There is no doubt that in this patient epinephrine was a valuable adjunct to miotic therapy. Many of the rises in tension pictured on the tension curve are attributable to failure to use all the drops on schedule.

In the last few years Diamox has had a very beneficial effect on the tension. Again

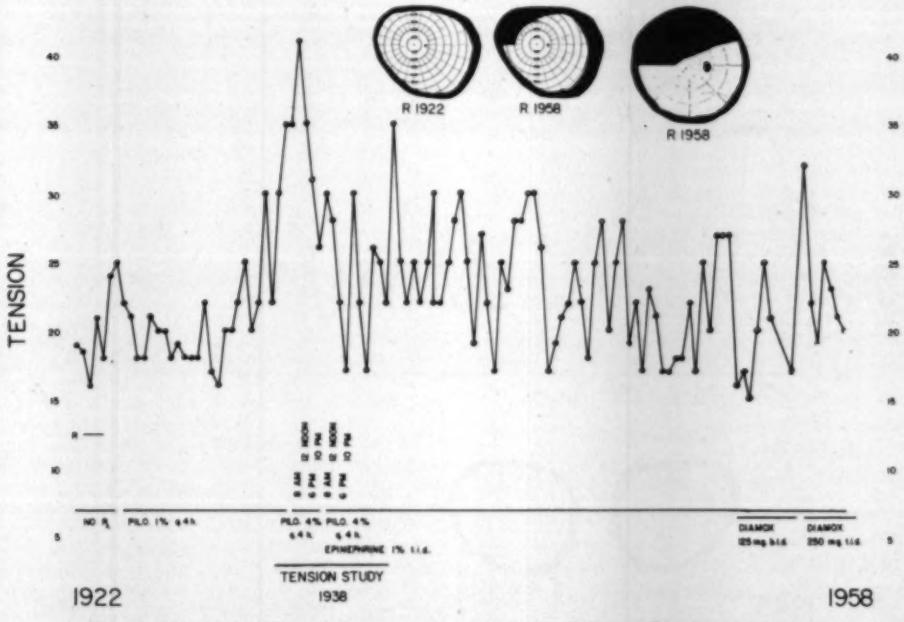

Fig. 11 (Chandler). Medical treatment. Average tension, 23.8 mm. Hg. Small field loss. Period of observation, 36 years.

most of the tension elevations in the last few years have been due to lapses in treatment. This patient still carries on his activities as an investment broker. He is aware that cataract extraction can be done when he feels sufficiently handicapped. Thus far neither the patient nor the surgeon has shown great enthusiasm for cataract extraction.

CASE 12.

This man at the age of 50 years was first examined in December, 1945. He had been under treatment for glaucoma for two or three months. Corrected vision in each eye was 6/5. Discs were normal, fields full. Tension was: R.E., 25 mm. Hg; L.E. 27 mm. Hg. The angle was narrow but open in both eyes. A tension study was carried out (fig. 12). It can be seen that one-percent and two-percent Pilocarpine failed to control the tension at all times during the day, but with four-percent Pilocarpine tension was normalized in both eyes. The patient was therefore continued on four-percent Pilocarpine every four hours.

As can be seen from the tension curve, tension remained generally in the low 20's or less. In 1957, all treatment was discontinued for 48 hours in order to allow the tension to rise and carry out gonioscopy with an elevated tension, to see if the angle was closed when the tension was elevated, since the angle was quite narrow. In this examination the angle appeared open except for one or two small areas which were questionable. This examination confirmed the diagnosis of open-angle glaucoma. Treatment was resumed and tension has continued at about the same levels. Corrected vision in 1958 is 6/5 in each eye. There is no pathologic cupping and the fields remain full.

Comment. This case illustrates the value of tension studies at the outset of treatment in determining the strength of miotic necessary to control the tension. It was obvious from this study that four-percent Pilocarpine was necessary at the outset, in this case, to control the tension. This case also demonstrates the fact that where there is no cupping of the discs, a reasonable control of tension may preserve the function of the eye over a period of many years. It can be noted the average tension in this case was in general

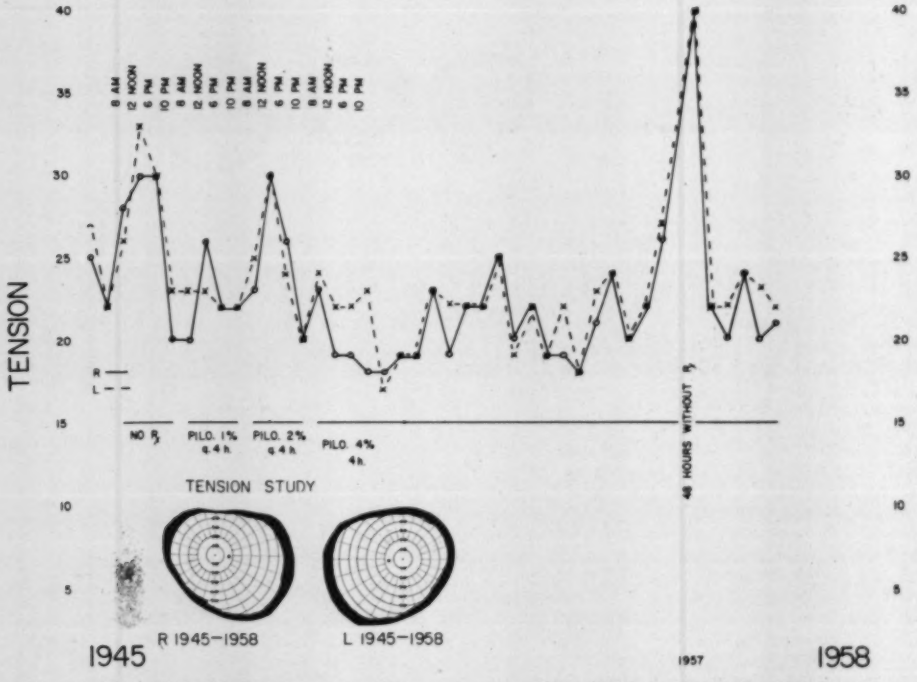

Fig. 12 (Chandler). Medical treatment. Average tension: R.E., 20.5 mm. Hg; L.E., 21.6 mm. Hg. No field loss. Period of observation, 13 years.

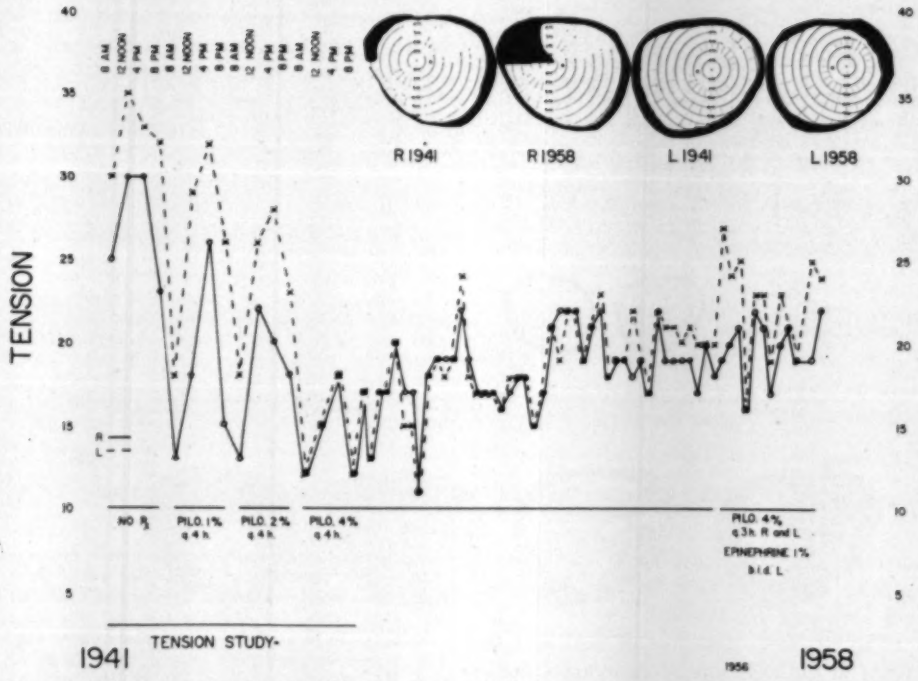

Fig. 13 (Chandler). Medical treatment. Average tension: R.E., 18.4 mm. Hg; L.E., 19.2 mm. Hg. Slight field loss in right eye, questionable loss in left eye. Period of observation, 17 years.

somewhat higher, 20.5 mm. Hg in the right eye, 21.6 mm. Hg in the left, than in some other cases of advanced glaucoma herein reported in which there was progressive loss of field over a period of years.

CASE 13

This man was first examined in 1941 at the age of 58 years. In the right eye corrected vision was 6/9. Tension was 41 mm. Hg. There was shallow cupping with some atrophy of the lower pole of the disc, and visual field study showed a small upper nasal step (fig. 13). In the left eye corrected vision was 6/5. Tension was 49 mm. Hg. The disc was normal and the field full.

A tension study was carried out over a period of four days, the tension being measured four times during the day. As can be seen from the tension chart (fig. 13), there was marked fluctuation of the tension during the day, with the highest reading in the middle of the day. It can be noted that one-percent Pilocarpine had very little effect, and even two-percent Pilocarpine lowered the tension only 7.0 or 8.0 mm. Hg. However when four-percent Pilocarpine was employed there was a striking drop in tension, with the highest reading 18 mm. Hg. Four-percent Pilocarpine was therefore prescribed for continuous use.

As can be seen in the tension curve, tension has been maintained at a satisfactory level during the ensuing 17 years. In the last few years the level of tension has gradually become higher. During the past two years, four-percent Pilocarpine has been used every three hours and 1.8-percent epinephrine bitartrate in the left eye twice a day. The field in the left eye has remained full except for a possible slight constriction nasally. There has been a gradual loss in the upper nasal field in the right eye. In 1958 corrected vision was 6/9 in the right eye, 6/6 in the left.

Comment. The striking feature in this case is the marked fluctuation of the tension during the day, the relative ineffectiveness of one- and two-percent Pilocarpine, and the marked effect of four-percent Pilocarpine. The tension study at the outset of treatment was very valuable in indicating that four-percent Pilocarpine was necessary to control the tension. It was therefore used from the beginning. As in many other cases, the oc-

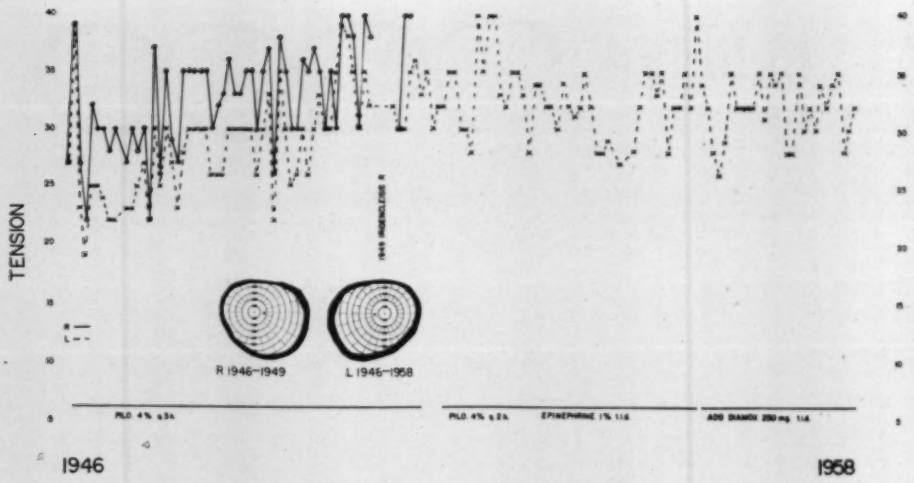

Fig. 14 (Chandler). Medical treatment. Average tension, 31.2 mm. Hg. No field loss. Period of observation, 12 years.

casional moderate elevations in tension were usually due to the fact that the drops had not been used on schedule.

The relatively small loss of field over the years in the right eye, demonstrates that a disc with limited cupping withstands pressure better than a markedly cupped disc, and the preservation of a practically full field in the left eye, that a normal disc is even more resistant to increased pressure.

CASE 14

This woman was first seen in 1946 at the age of 66 years. She had been under treatment for glaucoma for one year. Corrected vision in the right eye was 6/5, in the left eye 6/6. Discs were normal, fields full. Tension was right and left 27 mm. Hg. Four-percent Pilocarpine every three hours was prescribed. Tension ranged from 23 to 30 mm. Hg in the left eye, from 30 to 35 mm. Hg in the right eye (fig. 14).

In 1949, tension in the right eye rose to 40 mm. Hg. An iridencleisis with sclerectomy was done on the right eye. Following operation the anterior chamber remained flat and the cornea edematous throughout. On the fifth postoperative day the anterior chamber was reformed by posterior sclerotomy for release of the subchoroidal fluid and air injection into the anterior chamber. The cornea remained edematous throughout, and eventually painful bullous keratitis developed. The cornea was curetted, and cauterized with 25-percent trichloracetic acid. This resulted in a quiet and comfortable eye with a nearly opaque cornea, and vision reduced to perception of hand movements.

During the next eight years the left eye was treated with four-percent Pilocarpine every three hours, later every two hours. Epinephrine bitartrate was added later and Diamox for the past three years. As can be seen from the tension curve (fig. 14) tension has generally ranged from 30 to 35 mm. Hg, at times reaching 40 mm. Hg, with an average tension of 31.2 mm. Hg. Nevertheless, the disc has remained normal and the field full. There is now some nuclear sclerosis but corrected vision is still 6/6.

Comment. In retrospect, both the patient and the surgeon regret having undertaken operation on the right eye. I have not seen such a disastrous effect on the cornea from a flat anterior chamber in any other case. In view of the preservation of function in the left eye in spite of a relatively high tension, one must conclude that the right eye might have done as well under medical treatment, though the tension was generally higher in this eye. Had loss of function occurred it might well have been slow.

This case illustrates the fact that, at least in some eyes, if the disc is normal, a tension in the low 30's may be tolerated over a period of many years without loss of function. The average tension in this eye is 31.2 mm. Hg. Needless to say, neither the patient nor the surgeon has considered operative intervention on the left eye. There is actually no

reason for operation in open-angle glaucoma as long as the disc is normal and the field full, except perhaps in the case of patients under 40 years of age with a considerably elevated tension.

Group III

Case 15

This man at the age of 68 years was first seen in February, 1948. Corrected vision in the right eye was 6/15, due at least in part to a macular lesion. Corrected vision in the left eye was 6/9. There was total cupping of both discs with considerable atrophy. Tension was: R.E., 21 mm. Hg; L.E., 19 mm. Hg. There was marked field loss in both eyes (fig. 15). Pilocarpine (two percent) was prescribed but it had little effect on the tension.

Seven months later there appeared to be further loss of field in the right eye and, in October, 1948 a trephining operation was done on this eye. There was never a good filtering scar postoperatively. For a few months tension in the right eye remained slightly lower than in the left but not significantly so. After a few months, there was no evidence of filtration at the site of operation.

During the next several years treatment consisted of four-percent Pilocarpine every four hours, later every two hours. In 1952, epinephrine bitartrate (1.8 percent t.i.d.) was added, and in 1956, Diamox. As can be seen in the chart (fig. 15), tension has usually ranged in the low 20's, sometimes a few points higher or lower. There has been gradual loss of field in both eyes. Vision in 1958 is: R.E., 6/60; L.E., 6/30. There is slight nuclear sclerosis in both eyes. The patient is now 78 years of age.

Comment. That this is true glaucoma is suggested by the occasional tension eleva-

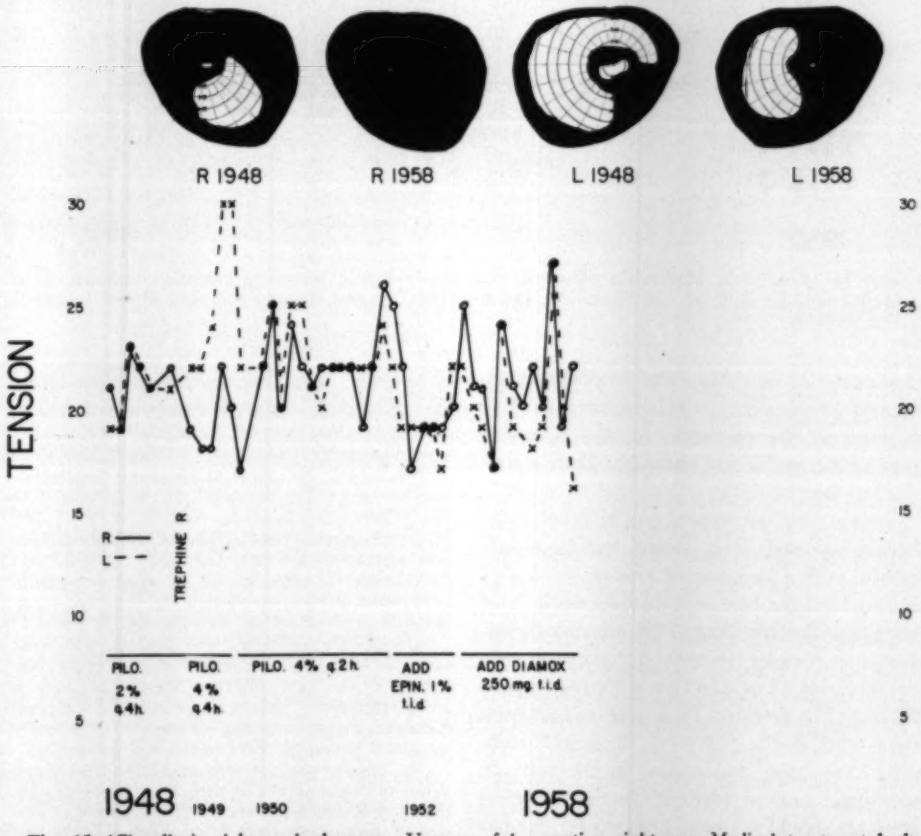

Fig. 15 (Chandler). Advanced glaucoma. Unsuccessful operation right eye. Medical treatment both eyes. Average tension: R.E., 21.3 mm. Hg; L.E., 21.5 mm. Hg. Progressive loss of field. Period of observation, 10 years.

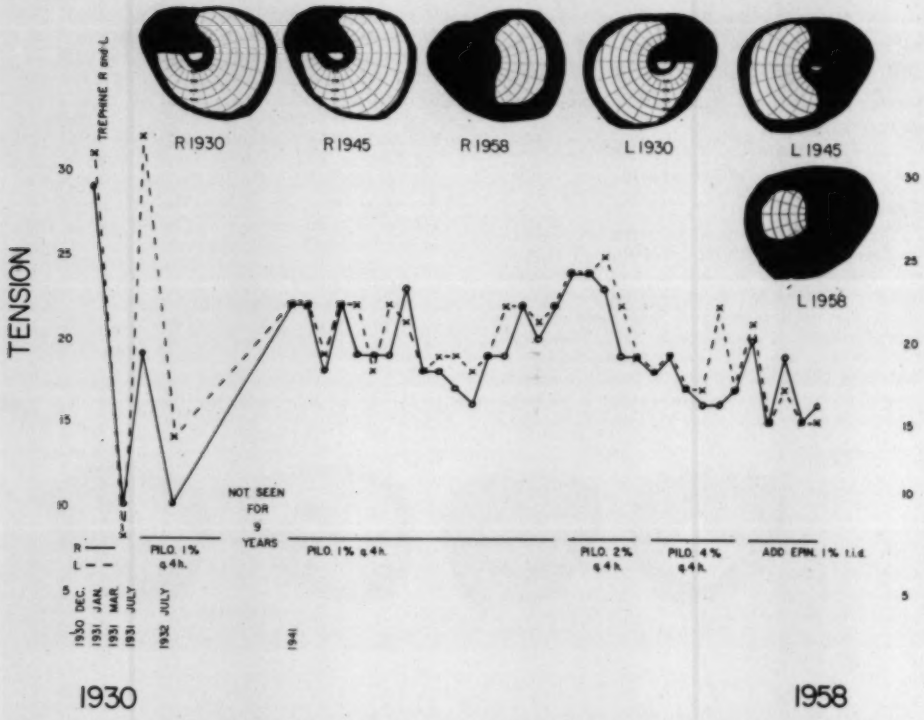

Fig. 16 (Chandler). Moderately advanced glaucoma. Partially successful operation. Average postoperative tension: R.E., 18 mm. Hg; L.E., 19.6 mm. Hg. Progress, slow loss of field. Period of observation, 28 years.

tions over 25 mm. Hg. The process was advanced when treatment was undertaken. Unfortunately the operation on the right eye was unsuccessful and there has been a continuing loss of field.

Judging by the experience in other cases herein reported, if operation had been successful with a lowering of tension to 15 mm. Hg or less, the rate of field loss might have been considerably slowed. In advanced glaucoma or pseudoglaucoma, I have not seen preservation of field in eyes with a tension of 20 mm. Hg or over. If operation had been successful in the right eye, it would probably have been undertaken in the left. In retrospect one wonders if further surgical attempts should not have been made. The patient's general health is now not good but, if he lives many more years, the prospects are great that he will become entirely blind.

CASE 16

This man at the age of 43 years was first examined by the late Dr. George S. Derby in December, 1930. Corrected vision was R.E., 6/12; L.E., 6/9. There was marked total cupping of both discs. Tension was: R.E., 29 mm. Hg; L.E., 31 mm. Hg. Field studies revealed loss of the upper nasal field, and a double Bjerrum scotoma in both eyes (fig. 16).

A trephining operation was done on both eyes early in 1931. Postoperatively though there was definite evidence of filtration there was never a really satisfactory bleb in either eye, and the postoperative tension was not as low as in a really successful operation. Pilocarpine was prescribed.

The patient was not seen again for nine years. When re-examined at this time, tension was 22 mm. Hg in each eye. One-percent Pilocarpine was prescribed. Since the patient was considerably myopic, stronger solutions caused marked blurring of vision.

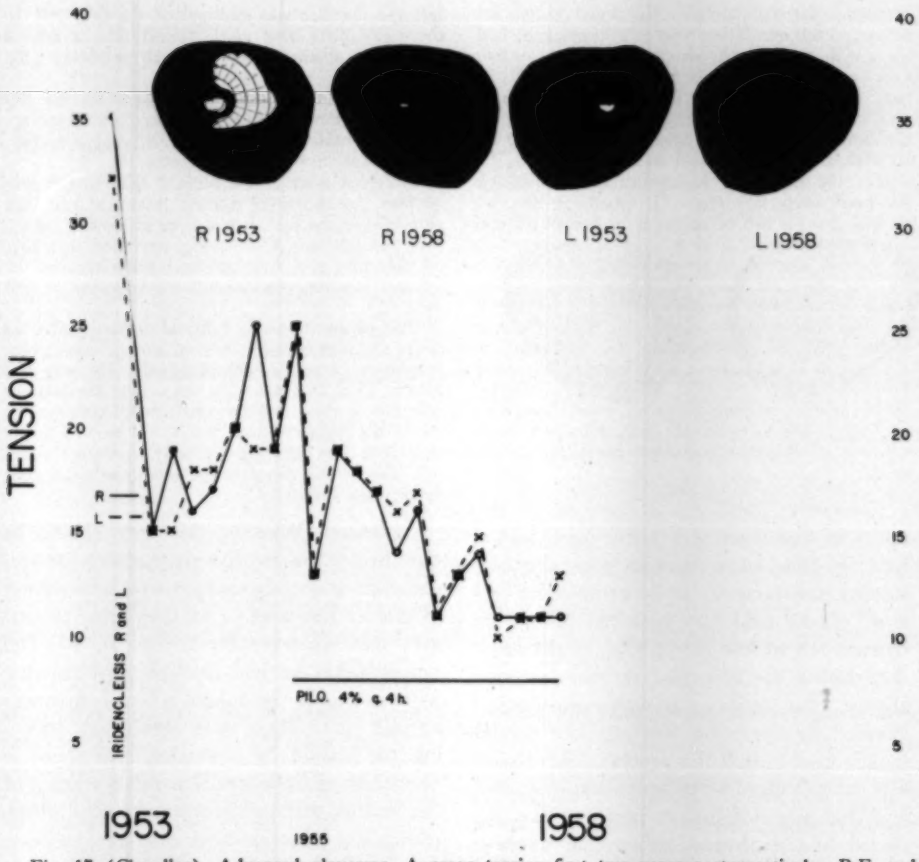

Fig. 17 (Chandler). Advanced glaucoma. Average tension first two years postoperatively: R.E. and L.E., 19 mm. Hg. During the next three years, R.E., 14 mm. Hg; L.E., 14.4 mm. Hg. Rapid progressive loss of field. Period of observation, five years.

During the next 17 years the patient was seen more or less regularly.

As can be seen from the tension curve (fig. 16), tension though in a range generally considered satisfactory, was not as low as one would wish for a case of moderately advanced glaucoma. Over the years, the peripheral fields have gradually shrunk. The central islands of vision were lost by 1953. There is now dense nuclear sclerosis in both eyes, and in 1958 vision in each eye is reduced to counting fingers at 1 foot.

Comment. This case is included to demonstrate that in advanced glaucoma if operation is only partly successful with a reduction of tension only to a level of approximately 20 mm. Hg one can usually not expect as good a control of the glaucoma as in cases where there is a good filtering scar and the tension postoperatively is 15 mm. Hg or less. There is generally a fairly good correlation between the level of tension and the progress of the disease. In general, the lower the tension, the more slow is further loss, if it occurs at all. Judging from the results of other cases in this series, if operation in this case had been completely successful, the loss of field might have been slowed or prevented.

CASE 17

This man was first seen in February, 1953, at the age of 70 years. He had been under treatment for

glaucoma for two months. Corrected vision was 6/9+ in each eye. There was total cupping of both discs with considerable atrophy. The angle in both eyes was wide and open. Tension was R.E., 35 mm. Hg; L.E., 32 mm. Hg. There was marked field loss in both eyes (fig. 17), the field in the left eye being reduced to a small central island. An iridencleisis was performed on each eye.

For the first year postoperatively tension was not particularly soft (fig. 17), usually 20 mm. Hg or less, but on two occasions a tension of 25 mm. Hg was recorded. Four-percent Pilocarpine was prescribed. The tension gradually fell to lower levels and for the past three years has generally been under 15 mm. Hg. Nevertheless, as can be seen in the field charts, this patient has became almost totally blind. In checking over the field charts in the records it is apparent that most of the loss of field had occurred by 1955 and therefore occurred during the period postoperatively when the tension was not as low as it became later. However, gradual loss of field has continued up to the present time.

Comment. This case is unusual in this series in that there was fairly rapid loss of field in spite of a tension generally considered satisfactory. It is noteworthy that most of the field loss occurred before the tension fell to low levels. One might argue that during the first year or two postoperatively, the tension though considerably lowered by operation was not low enough in this case to halt the process. The disease was far advanced when operation was done, and this may account in part for the continued loss. However, some other cases in this series were almost as advanced at the time of operation, yet maintenance of tension at 15 mm. Hg or less resulted in little loss of field over a period of many years. If the advanced state of the glaucoma at the time of operation is the important fact in the continued loss, it would provide a valid reason for earlier surgical intervention.

Group IV

Case 18

This man at the age of 47 years was first examined in 1930. He gave a history of having used drops in the right eye for eight years. No data were available as to the nature of the drops or the state of the visual field when treatment was begun eight years previously. At the first examination corrected vision was 6/5 in each eye. The right disc was deeply and totally cupped and atrophic. In the left eye the disc was normal and the field full. In the right there was marked field loss, a central island and a small temporal field remaining (fig. 18).

Slitlamp examination was negative in the left eye but, in the right, the posterior corneal surface showed considerable pigment dust, especially centrally. Tension was: O.U., 18 mm. Hg.

Since, considering the marked field loss, it was obvious that whatever medical treatment had been employed in the past, it had been ineffective, operation was advised. A trephining operation was done on the right eye. A large cystic filtering scar developed resulting in a significant lowering of tension.

The patient lived in England for a number of years and then in South Africa, so was seen at very infrequent intervals postoperatively. He was last seen in 1950, 20 years after operation. The left eye was still normal in all respects with a tension of 19. In the right eye there was still a broad filtering scar. The lens was grossly clear, corrected vision 6/9. The field in the right eye was exactly the same as 20 years earlier.

Comment. Whether this case should be classified as low-tension glaucoma or pseudoglaucoma is not of great practical importance. Whatever the level of tension prior to surgery and whatever the treatment, the fact remains that he had marked total cupping with extensive field loss when I first examined him. There was significant lowering of tension by operation and there is every reason to believe that this lowering of the tension was the important factor in arresting the process. This case can be compared with Cases 15 and 16 in which operation was unsuccessful in lowering the tension and field loss continued.

Case 19

This woman at 48 years of age came under the care of the late Dr. George S. Derby in 1916. The right eye was 14 diopters myopic, the left 13. Corrected vision was 6/9 in each eye. Suggestive cupping of the disc was noted in each eye. Tension was: O.U., 19 mm. Hg. No treatment was employed until 1919, when one-percent Pilocarpine was prescribed with little if any effect on the tension. The first field was recorded in 1920 (fig. 19).

It can be noted that most of the nasal field had been lost in both eyes and there was an upper Bjerrum scotoma in each eye. It can be seen in the chart that preoperatively the tension ranged from 16 to 19. In 1920 a trephination was done on the right eye by Dr. Derby, and in 1921 on the left eye. A good filtering scar developed in both eyes. By 1924 the filtering scar had closed in the left eye and ten-

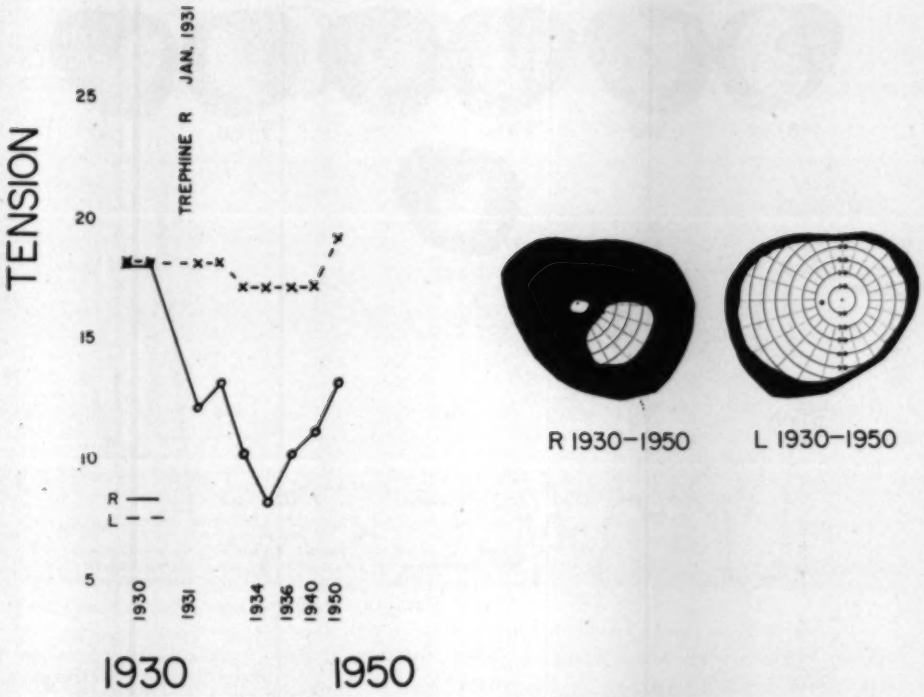

Fig. 18 (Chandler). Probable pseudoglaucoma with marked field loss. Average postoperative tension, 11 mm. Hg. No field loss. Period of observation, 20 years.

sion had risen. Another trephination was done on the left eye in 1925 by Dr. Derby, with a good filtering scar resulting.

The filtering scar in each eye continued to function with maintenance of an average tension of 9.6 mm. Hg in the right eye and 11.5 mm. Hg in the left eye until 1953 when a cataract extraction was done on each eye. Thus the trephination continued to be effective in the right eye for 33 years, and the second trephination in the left eye for 28 years.

Fields were recorded once or twice a year in the right eye up to 1938, and in the left eye up to 1945. Field tests were discontinued at these times when increasing lens opacities made perimetry impractical. It can be seen that there was little loss of field over the years.

By 1953, there had been a mature cataract in the right eye for a number of years and the lens opacities in the left had increased to the point where vision was reduced to counting fingers at two feet. Cataract extraction had been deferred according to the wish of the patient.

In 1953 an uneventful intracapsular cataract extraction was performed on each eye. Resultant vision was at first 6/60 in the right eye, 6/30 in the left, due to myopic macular changes. Unfortunately, shortly after cataract extraction the filtering scars began to close in both eyes with a resultant elevation of tension which could not be effectively controlled with medical treatment. Vision gradually failed and just before the patient died in 1956 at 88 years of age, vision was reduced to counting fingers at six feet in the right eye, at one foot in the left.

Comment. That this is a case of true glaucoma with a relatively low tension is suggested by the loss of field which had occurred preoperatively, the prevention of further loss of field by lowering the tension by operation, and the continuing loss of vision after cataract extraction, when the filtering scars partially closed and the tension again became higher. However, the elevated tension when the filtering scars partly closed following cataract extraction, could be explained by peripheral anterior synechias resulting from the previous trephining operations. One cannot be certain, therefore, that this was not a

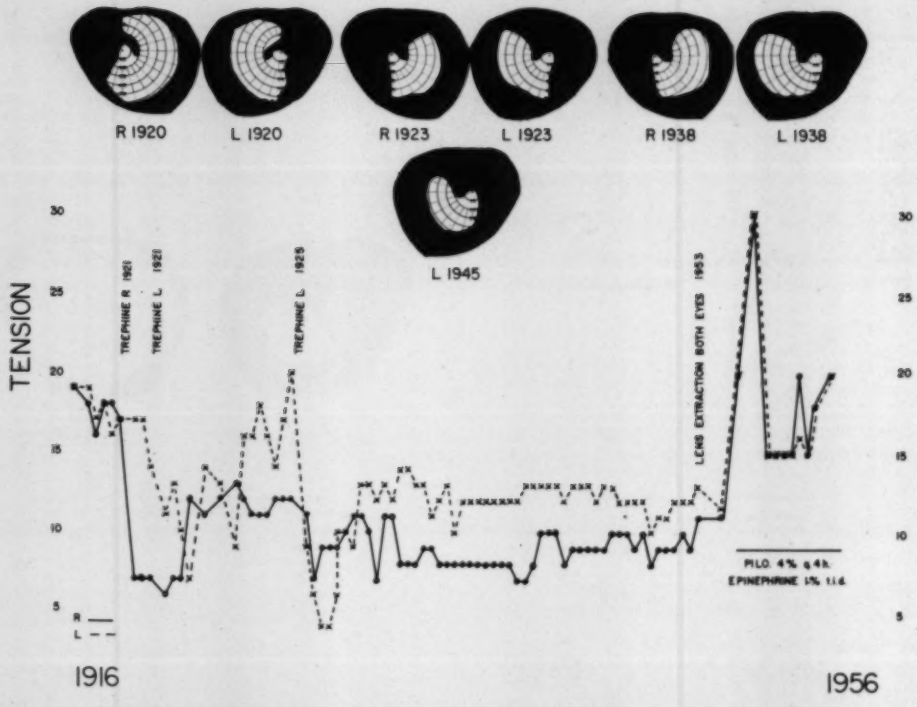

Fig. 19 (Chandler). Probable pseudoglaucoma with considerable field loss. Average preoperative tension: R.E., 17.5 mm. Hg; L.E. 17 mm. Hg. Average postoperative tension: R.E., 9.6 mm. Hg; L.E., 11.5 mm. Hg. Moderate field loss postoperatively. Period of observation, 40 years.

case of so-called pseudoglaucoma. The condition was probably pseudoglaucoma in the beginning, real glaucoma due to peripheral anterior synechias, when the filtering scars partly closed following cataract extraction.

The average preoperative tension in the right eye was 17.5 mm. Hg, postoperative 9.6 mm. Hg; in the left eye 17 and 11.5 mm, Hg respectively. This lowering of the tension from the normal to a somewhat subnormal range was apparently sufficient in these vulnerable eyes to halt the loss of function. There is every reason to consider that without operation this patient would have been blind many years ago. All other patients I have observed with such advanced field changes when first examined, have steadily lost field over a period of years when the tension remained 17 to 20 mm. Hg. Doubtless few ophthalmologists would have had the courage to undertake operation in such a case in 1920.

CASE 20

This woman was first seen in 1934 at the age of 44 years. Corrected vision was 6/5 in each eye. Tension was right and left 15 mm. Hg. There was cupping of the lower outer border of the disc in the right eye, a small field defect consisting of a 10 degree upper nasal step and a narrow Bjerrum scotoma (fig. 20). The left disc was normal and the field full.

A tension study was carried out (fig. 20). It can be seen that the tension in the right eye was never over 17 mm. Hg during the day or night, generally somewhat lower than in the left eye. During the next several months there was a definite increase in the size of the Bjerrum scotoma, and a slight increase in the nasal step. A trephining operation was done on the right eye.

Tension over the next 24 years is depicted in the chart. For several years tension in the right eye was 3.0 or 4.0 mm. Hg lower than in the left.

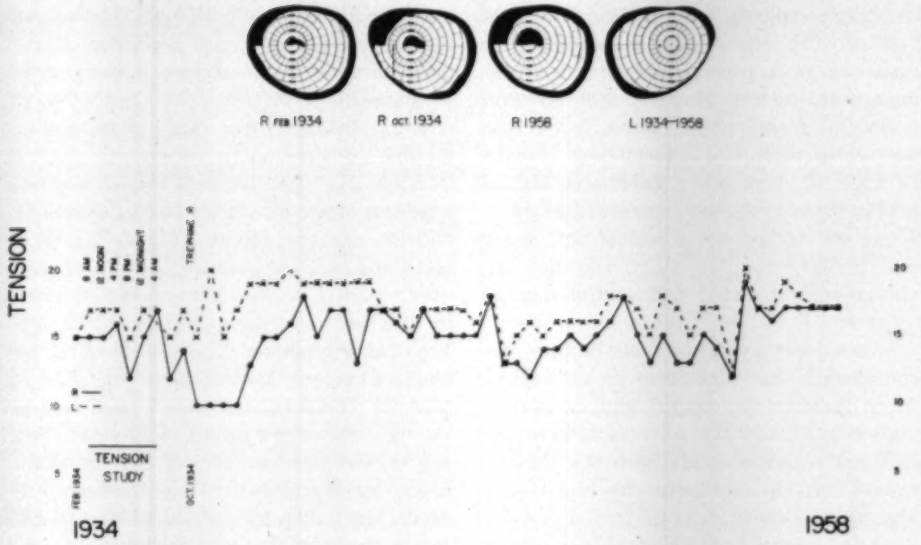

Fig. 20 (Chandler). Pseudoglaucoma. Operation on right eye only. Average postoperative tension, 14.8 mm. Hg. L.E., untreated, average tension, 17.2 mm. Hg. Slight loss of field, right eye. No field loss, left eye. Period of observation, 24 years.

It has continued to be slightly lower, though for a number of years there has been very little difference in the tension of the two eyes.

In the 24 years since operation there has been a slight loss of field in the right eye. The field in the left eye has remained full. The lower outer pole of the left disc now shows cupping but there is no atrophy. There is slight nuclear sclerosis in both eyes, but corrected vision is 6/6 in each eye.*

In 1952 gonioscopy revealed a normal open angle in the left eye. In the right eye there were a certain number of peripheral anterior synechias some of which encroached on the corneoscleral meshwork, evidently the result of operation.

Tonography by Dr. W. M. Grant showed a facility of outflow of 0.26 in the right eye, 0.27 in the left eye. Since some of the corneoscleral meshwork in the right eye was blocked by peripheral anterior synechias, it would appear that the trephine opening was still functioning at least to some extent.

Comment. Since the facility of outflow was quite normal in the left eye, one might well assume that it would have been equally good in the right eye prior to operation. It therefore appears that this was not a case of true glaucoma. However, the field defect and its progression in the right eye and the development of cupping of the left disc certainly simulates glaucoma. Though some lowering of the tension resulted from operation on the right eye, it was not significant in extent. One might well question whether operation was justified and whether it had any effect in slowing the process. With the relatively low tension preoperatively one might have expected the loss of field to be slow even without operation. The definite loss of field noted preoperatively and the relative youth of the patient made operation seem justified at the time.

* In January, 1959, the left eye became entirely blind from occlusion of the central retinal artery.

General comment

In four groups of cases the results of long-term therapy of true glaucoma and of so-called pseudoglaucoma have been presented.

In studying the data in group I, certain facts may be noted. It is evident that if operation is completely successful with tension postoperatively maintained at a sufficiently low level, further loss of field may

be either completely prevented or the rate of loss may be considerably slowed over a period of many years. In the first five cases there was no loss of field whatever over periods of from 14 to 27 years. In the next four cases there was a small loss of field. In Case 10 there was considerable loss of field in the left eye over a period of 31 years. There was no instance of loss of field in any case in this group in which the disc was normal and the field full at the time of operation.

It is noteworthy that in Case 10, there was considerable loss of field in the left eye in which the disc was cupped, with an average tension of 15 mm. Hg, whereas there was no loss in the right eye in which the disc was normal, with an average tension of 16.6 mm. Hg. The continued loss of field in the left eye over a period of 30 years in spite of a consistently low tension is exceptional in this series. It suggests that there may be other factors in this case in addition to the tension per se which played a part in the loss of field. However, there is every reason to believe that without the low tension brought about by operation this eye would have been blind many years ago. The right disc was normal and the field full at the time of operation whereas the left disc was cupped and there was a considerable field defect at the time of operation.

It is remarkable that in Case 8, the tiny central field has been maintained over a period of 31 years, in spite of an average tension of 19 mm. Hg. This case is exceptional in this series. In all other cases of advanced glaucoma where tension was at corresponding levels, there was progressive loss of field as illustrated by the cases in Group III.

In Group II where only medical treatment was employed, with the exception of the right eye of Case 13 which showed limited cupping of the lower pole of the disc, all discs were normal when treatment was commenced. It can be seen that the average tension in this medically treated group was considerably higher than in Group I. The results in this group demonstrate again that a normal disc withstands increased pressure relatively well.

It can be noted that in the right eye of Case 7, in spite of an average tension of 24.5 mm. Hg, there has been loss of only one quadrant of the field in 32 years. In Case 11 with an average tension of 23.8 mm. Hg, there has been no considerable loss of field over a period of over 30 years. In Case 14, in spite of an average tension of 31.2 mm. Hg over a period of 12 years, the field remains full. It is interesting to note that in Case 13 there has been some loss of field in the right eye over a period of 17 years. This eye showed some cupping of the disc at the beginning of treatment. There has been little or no loss in the left eye in which the disc was normal, yet the average tension in the left eye was 19.2 mm. Hg, slightly higher than in the right in which the average tension was 18.4 mm. Hg.

The cases in this group demonstrate well the fact that a normal disc withstands increased tension relatively well. It is probably true that all normal discs do not withstand increased pressure equally well. In my experience, the rosy disc with prominent blood vessels commonly seen in high hyperopia appears to tolerate increased tension better than the type of disc usually seen in high myopia, and discs with wide, deep physiologic cups appear to be more vulnerable than those with small shallow cups. There may be other factors which influence the ability of the normal disc to tolerate increased tension. However, if in a certain individual the disc in one eye shows pathologic cupping and in the fellow eye only physiologic cupping, one will usually find that a level of tension which may be well tolerated by the eye with the normal disc may cause progressive loss of field in the eye with pathologic cupping.

Cases 11, 12 and 13 illustrate the value of tension studies in determining at the outset the most effective treatment in lowering the tension. In Case 11 the addition of 1.8-per-

cent epinephrine bitartrate to treatment with four-percent Pilocarpine produced a striking further lowering of the tension. In Cases 12 and 13, one- and two-percent Pilocarpine were relatively ineffective, whereas four-percent Pilocarpine brought about complete normalization of tension.

In Group III, where operation was unsuccessful or only partly successful, in all cases the discs were totally cupped and the glaucoma was advanced when operation was done, far advanced in Case 17. In Case 15 there was no lasting benefit from operation. In Case 16, though the tension was reduced by operation, it was not brought to the low levels of most of the cases in Group I.

In Case 17, the average tension for the first two years postoperatively was 19 mm. Hg, and during this period there was considerable loss of field. During the next three years the average tension was low—14 mm. Hg in the right eye, 14.4 mm. Hg in the left, yet gradual loss of field continued and this patient is almost blind. This case is exceptional in my experience. Though to be sure the disease was far advanced at the time of operation, in five years this patient became almost totally blind, in spite of what must be considered a satisfactory tension level at least during the past three years. As in Case 10, there may have been other factors at work in this case in addition to the tension per se.

In the light of the results in several cases in Group I, it would appear that in the cases in Group III, if operation had been completely successful and the tension brought promptly to really low levels, the progress toward blindness might have been considerably slowed or possibly prevented entirely.

The cases in Group III illustrate the fact that if the disc is totally cupped, the eye withstands pressure poorly. This is particularly evident when one compares the cases in this group with those in Group II.

The cases in Group IV probably do not represent instances of true glaucoma, but rather of so-called pseudoglaucoma or cavernous atrophy. In these cases typical glaucomatous type cupping of the discs is seen and glaucomatous types of field defects, with the tension always well within the normal range. In Case 20, tonography on the unoperated eye showed a completely normal facility of outflow.

Cases of this type are certainly not rare in general ophthalmologic practice. In most cases the condition develops past middle life. The incidence appears to become higher with every increasing decade of life. The loss of field is progressive, but generally slow, and rarely leads to blindness. In this group all the patients were in the forties when operation was done.

There was significant lowering of tension by operation in Cases 18 and 19, and though there was marked cupping and rather advanced field loss at the time of operation, there was little or no further loss of field over a period of 20 to 30 years. In Case 19 when, after cataract extraction, there was partial closure of the filtering scars and tension rose above 20 mm. Hg, there was steady progression to almost complete blindness in a period of three years. In Case 20, operation must be considered only partly successful, since the postoperative tension was not as low as in the other cases, and there was continued slow loss of field.

The experience with these and similar cases with a shorter period of follow-up suggests that, regardless of whether one is dealing with true glaucoma or with pseudoglaucoma, if by operation tension can be brought to a sufficiently low level, the loss of function may be arrested. One would probably never undertake operation in patients 50 or more years of age with this condition, since the loss of field is generally slow and, if there is relatively little loss of field at the first examination, few of these patients will become entirely blind. However, if there is marked field loss at the first examination, operation is to be considered if there is progressive further loss, even in patients past 50 years of age. The evidence

strongly suggests that in Cases 18 and 19 the condition would have steadily progressed without operation.

One cannot lay down hard and fast rules for the management of glaucoma. There are exceptions to all rules. Nevertheless, from a study of the cases presented here as well as experience with other cases, certain broad conclusions seem justified.

Summary and Conclusions

1. Twenty cases illustrating the results of long-term treatment of glaucoma are presented. Ten with successful operations, four treated medically, three showing the results of unsuccessful or partly successful operation and continued medical treatment, and three of surgical treatment of probable so-called pseudoglaucoma.

2. It is concluded that eyes with advanced glaucoma, in which there is marked cupping of the disc and considerable field loss, withstand increased tension poorly and require a tension below the average normal to prevent further loss of function.

3. Eyes with limited cupping, confined to one pole of the disc, appear to withstand increased tension considerably better than eyes with total cupping of the disc.

4. Eyes with a normal disc and no loss of field appear to withstand increased pressure well and may show no loss of function over a period of many years with a tension at levels which would cause progressive loss of field in eyes already showing cupping of the disc.

5. Whatever the mechanism of cupping and atrophy of the optic disc, it appears that the ophthalmoscopic appearance of the optic disc may serve as an important guide in the management of glaucoma. A certain level of tension may have quite a different significance, depending on whether the disc is normal, or shows limited or total cupping. If the disc is normal, it is evident that some eyes may tolerate a tension in the low 30's for many years without loss of function, whereas for eyes with considerable cupping, such a tension would be far too high.

6. Whatever the stage of the disease, if operation is successful in reducing the tension to a low enough level, there is an excellent chance of preventing further loss of function.

7. In cases of pseudoglaucoma showing progressive loss of field, operation, if successful in bringing about a significant lowering of tension, appears to have a very favorable influence in arresting the process. Operation therefore appears justified for some patients showing progressive loss of field.

8. In the medical treatment of glaucoma, tension studies are of considerable value as a means of discovering quickly what treatment is most effective.

5 Bay State Road (15).

CONJUNCTIVOANTROSTOMY*

A surgical treatment of epiphora

A preliminary report

James E. Bennett, M.D., James R. Armstrong, M.D., and Albert G. Schraff, M.D.
Cleveland, Ohio

Introduction

Epiphora has always been a challenging problem to the ophthalmic surgeon. The epiphora resulting from occlusion of the nasolacrimal duct has been successfully corrected by dacryocystorhinostomy, which has become the classical operation of lacrimal surgery. However, the epiphoras resulting from abnormalities or stricture peripheral to

* From the Department of Medicine and Surgery, Section of Ophthalmology, Crile Veterans Administration Hospital, Cleveland, Ohio.

the lacrimal sac have presented a much more challenging problem, without a completely satisfactory solution. Many operations have been devised for the treatment of epiphora, each being adapted to a specific problem. There are many excellent texts and review articles discussing these procedures, therefore, they will not be reviewed nor discussed here.

Conjunctivoantrorhinostomy is a gravity drainage procedure, utilizing the maxillary sinus, which was designed and has been demonstrated to control successfully the extensive epiphora following parotid duct transplantation. This operation has been modified and adapted for use in epiphora resulting from abnormalities or strictures of the peripheral nasolacrimal drainage system.

Discussion

Conjunctivoantrostomy has been designed to provide gravity drainage of the lacus lacrimalis completely independent of the normal lacrimal drainage system. It consists of making an opening from the inferior conjunctival cul-de-sac into the maxillary sinus, as far medially as possible, thus allowing a free gravity drainage of tears from the lacus lacrimalis into the maxillary antrum. Drainage from the maxillary antrum into the nose is accomplished through the normal ostia of the maxillary sinus.

Conjunctivoantrostomy, being completely independent of the normal lacrimal drainage system, is therefore applicable to any case of epiphora. Previously unsuccessful lacrimal surgery does not preclude the use of conjunctivoantrostomy. It is particularly adaptable to epiphora resulting from strictures, abnormalities, or absence of the lacrimal puncta or canaliculi or following dacryocystectomy.

Surgical procedure

Under general anesthesia, a conjunctival incision is made in the medial one third of the conjunctival cul-de-sac and carried down to the bony rim of the orbit. The periosteum is elevated, baring the bone over the orbital rim, thus allowing a one-cm. bony opening to be made into the maxillary sinus. This opening must be made as far medially as possible to allow a vertical drainage path from the lacus lacrimalis into the maxillary antrum.

After creation of the bony opening, the antral mucosa is excised in the area of the opening. Triangular conjunctival flaps are then prepared, with the base of one flap being medial so as to create a vertical funnel from the lacus lacrimalis. The conjunctival incision must be made deep in the cul-de-sac to prevent traction on the lid and subsequent entropion.

A 4-0 black silk suture is passed through the apex of each flap to be used to draw the conjunctival flaps down to line the bony opening. These sutures are then passed through the lumen of a large-caliber polyethylene tube, which is then inserted through the bony opening and securely taped to the forehead. Traction can then be adjusted individually on the black silk sutures, drawing the conjunctival flaps down to line satisfac-

Fig. 1 (Bennett, Armstrong and Schraff). Roentgenogram of lipiodal injection of canaliculi, illustrating stricture of the canaliculi just prior to the entrance into the lacrimal sac.

Fig. 2 (Bennett, Armstrong and Schraff). Photograph showing conjunctivoantrostomy opening four months after surgery.

torily the bony opening and to create a vertical funnel. No dressing is necessary.

The polyethylene stent is left in place for seven days and is then removed. The black silk sutures have sloughed free by this time and are removed. During the postoperative period, the maxillary sinus can be freely irrigated through the polyethylene stent. Daily irrigations are advisable postsurgically to remove any blood that may have accumulated in the sinus. No stent is subsequently required.

Case report

A 47-year-old white man was admitted to the eye service at Crile Veterans Administration Hospital complaining of epiphora of the left eye of two years' duration. Irrigation of the lacrimal passageways was unsuccessful, and no return occurred through either canaliculus. Lipiodal injection of both the left upper and lower canaliculi was performed (fig. 1) and revealed a stricture at the junction of the superior and inferior canaliculi. Attempts to pass a probe past this stricture were unsuccessful.

On May 12, 1959, a conjunctivoantrostomy was performed by the previously described surgical technique. The conjunctivoantrostomy opening has remained patent and functional without the use of

Fig. 3 (Bennett, Armstrong and Schraff). Roentgenogram showing maxillary sinus to be clear four months after conjunctivoantrostomy. No nasoantral window was made, drainage occurring through the normal ostia. The bony defect in the medial portion of the anterior rim of the orbit can be visualized.

a stent (fig. 2). Postoperative infection has not occurred. The maxillary sinus has remained clear by X-ray films, although no nasoantral window was made (fig. 3). The epiphora has been effectively controlled.

Summary

The successful treatment of a case of epiphora secondary to strictures of the lacrimal canaliculi by conjunctivoantrostomy is reported. The surgical technique is described. Conjunctivoantrostomy provides a purely gravity drainage system utilizing the maxillary sinus which is completely independent of the normal lacrimal drainage system. Thus, it would seem to be applicable to epiphora following dacryocystectomy, or from strictures, abnormalities, or absence of the lacrimal puncta or canaliculi.

Conclusions

Conjunctivoantrostomy has been demonstrated to be a feasible surgical procedure for the treatment of epiphora. The extent of its application awaits further clinical evaluation.

5500 Ridge Road (29).

Reference

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ULTRASOUND IN OPHTHALMOLOGY*

Gilbert Baum, M.D.
Port Chester, New York
and
Ivan Greenwood,† B.S.
Pleasantville, New York

Visualization of the interior of the light-opaque eye and orbit is the most promising ophthalmic application of ultrasound. As a diagnostic tool, its potential value compares with that of the ophthalmoscope.

By means of ultrasound, retinal detachments, tumors, and radiolucent foreign bodies may be visualized despite a light-opaque cornea, lens and vitreous. A differential diagnosis among vitreous hemorrhages, retinal detachment and tumors may also be made despite the presence of opacification to light. Radiolucent foreign bodies within such hemorrhages, orbital lesions, and tumors of the macula have also been demonstrated with this technique.

Such demonstrations have been accomplished by applying sonar and radar techniques to tissue. Sonar operates by sending forth repeated bursts of high frequency sound, that is, greater than 20,000 cycles per second. The sound waves strike the tissues

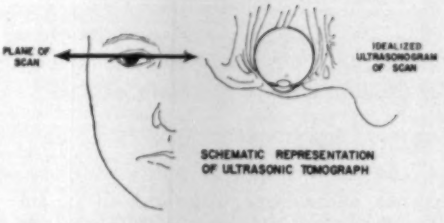

Fig. 1 (Baum and Greenwood). Schematic representation of an ultrasonic tomogram. Serial tomography is performed by scanning eye at successive levels.

* From the Department of Ophthalmology, New York University Post-Graduate Medical School, the Bronx Veterans Administration Hospital.

† Assistant Director of Research, General Precision Laboratory, Inc.

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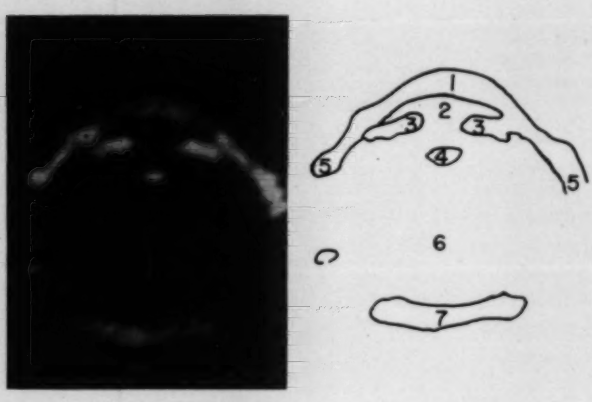


Fig. 2 (Baum and Greenwood). Ultrasonogram of a human eye. (1) Cornea. (2) Anterior chamber. (3) Iris. (4) Lens. (5) Anterior sclera. (6) Vitreous. (7) Posterior sclera. All portions of the eye can be visualized by this equipment by having the patient look right and left. New equipment under construction will visualize the entire eye to the apex of the orbit in a single scan.

and are reflected. The echoes are sequentially received and presented on the cathode ray tube of a radar receiver. The physician observes the images of the interior structures of the body on the face of the tube. These images, or ultrasonograms, are cross sectional views, that is, they are compound coronal-saggital sections of the eye and orbit as viewed from an anteroposterior position (fig. 1). This equipment operates at 15 mc.

The face of the tube may be photographed to obtain permanent records of particular views, such as are presented in this paper (fig. 2).‡

Motion of the eyes is portrayed on the face of the cathode ray tube, much as motion of an actor is portrayed on a television set. Analysis of the motion of the tissues may facilitate diagnosis.[1]

Ocular applications

Retinal detachments, intraocular tumors, macular lesions, vitreous hemorrhages and radiolucent foreign bodies have been demonstrated by ultrasonography. The following cases illustrate such diagnoses.

Retinal detachment

The patient (fig. 3) had a semiopaque cornea, and a hemorrhage in both the anterior chamber and vitreous of the eye. The interior of this eye could not be visualized with any existing ophthalmic instrument. Ultrasonic visualization demonstrated both the intraocular pathology, and orbital structures. The former proved to be a total retinal detachment which contained within it the vitreous hemorrhage (fig. 4).

Serial ultrasonography (figs. 5, 6, and 7) demonstrated the shift in position of the detached retina at different levels through the eye. The diagnosis and location of the retina were confirmed by the pathologic sections (figs. 5P, 6P, and 7P).

Tumors of the eye

A diagnosis of melanoma of the ciliary body could be made in this eye only after extension of the tumor into the angle and iris had occurred, since the interior of the eye was obscured by a cataract (fig. 8). On the ultrasonogram the tumor is clearly visible

Fig. 3 (Baum and Greenwood). Gross appearance of patient's eyes. Note the haziness of the cornea and the hyphema of the right eye.

‡ A detailed discussion of the theory and operation of this instrument will be found in reference 1.

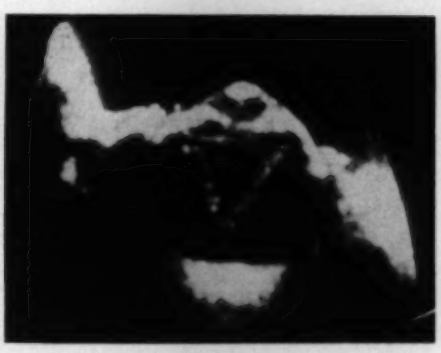
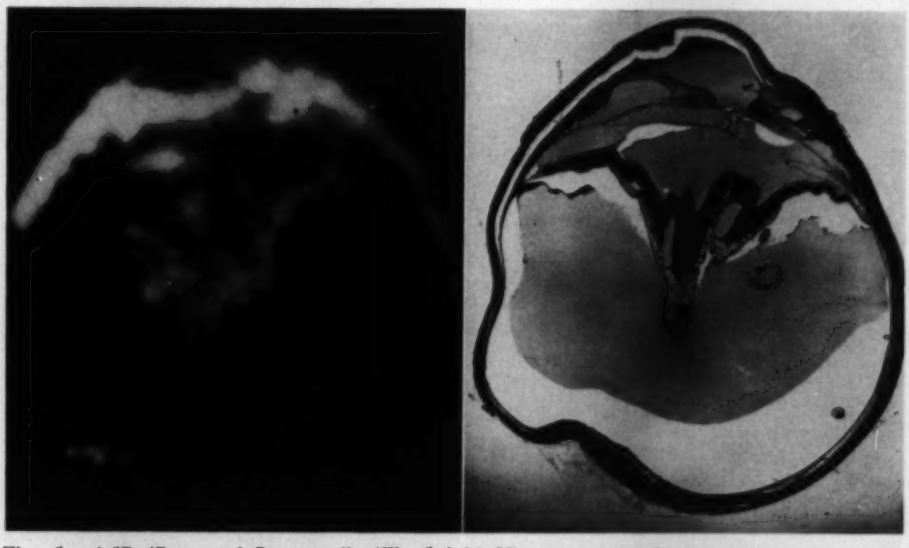
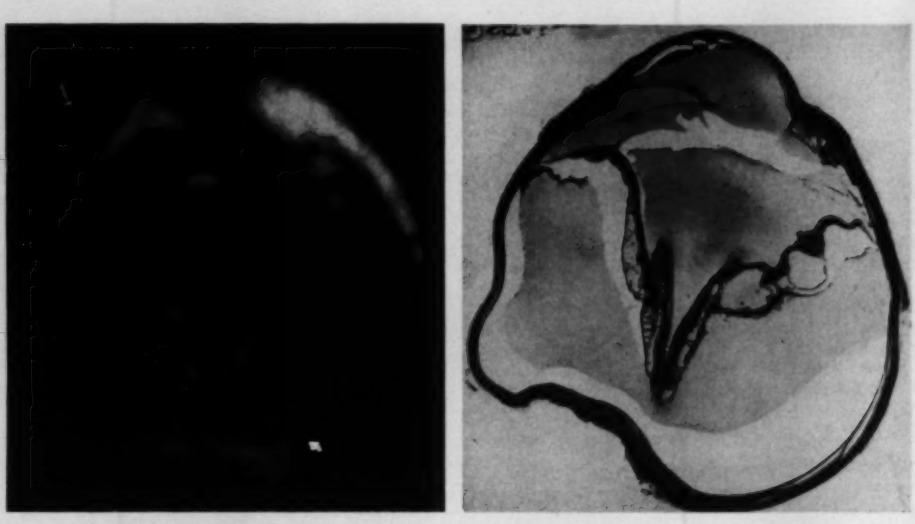


Fig. 4 (Baum and Greenwood). Echogram of right eye in situ of patient in Figure 3. (1) Nose. (2) Nasal cavity. (3) Nasolacrimal sac. (4) Caruncle. (5) Cornea. (6) Anterior chamber. (7) Ciliary body. (8) Iris. (9) Vitreous hemorrhage and bands. (10) Detached retinal. (11) Orbital fat. (12) Lateral rectus muscle at posterior pole of the eye.



Figs. 5 and 5P (Baum and Greenwood). (Fig. 5, left) Ultrasonogram* of enucleated eye of patient in Figures 3 and 4. (Fig. 5P, right) Tissue section through same level of eye.

* The sonograms shown in Figures 5, 6 and 7 are through different levels of the same enucleated eye.



Figs. 6 and 6P (Baum and Greenwood). (Fig. 6, left) Ultrasonogram* of same patient. (Fig. 6P, right) Tissue section through same level of eye.

behind the cataract and diagnosis could have been made at an earlier date (fig. 9). The diagnosis and location were confirmed by the histopathology section (fig. 10).

At present, tumor identification is based upon the appearance of an intense echo from an aberrant site, for example, a mass in the vitreous body. Interpretation is facilitated by

Figs. 7 and 7P (Baum and Greenwood). (Fig. 7, left) Ultrasonogram* of same patient as in Figures 3, 4, 5 and 6. (Fig. 7P, right) Tissue section through same level of eye.

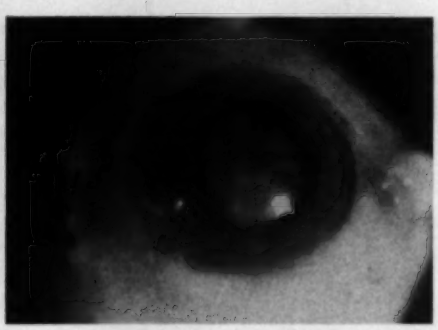


Fig. 8 (Baum and Greenwood). Gross appearance of an eye containing a melanoma of the ciliary body. (Patient of Dr. Algernon B. Reese.)

tracing the mass back to its base by serial ultrasonography and noting its site of origin. In addition, the mobility of the mass may be checked by having the patient move his eye while the ultrasonoscope is observed. Other phenomena such as the appearance of sonic vacuoles and shadows may also aid interpretation.

Macular lesions

Only the surface of lesions of the posterior segment of the eye can be seen with existing ophthalmic instruments. Cross sectional views of posterior lesions may for the first time be obtained by ultrasonography without enucleating and sectioning the globe. Though the present sonograms are not sufficiently clear to permit reliable diagnoses to be made of these conditions, a new instrument, presently under construction, will greatly improve the obtainable definition.

A fundus photograph of this eye illustrates the diagnostic problem presented by this case (fig. 11). Despite the small size of the macular lesion, approximately six-mm. diameter by one-mm. thickness, the lesion is readily discernible on the ultrasonogram (fig. 12). An area of central thinning within the lesion can be noted (arrow). Because of the lack of interpretive experience, this feature was missed prior to enucleation. An

Fig. 9

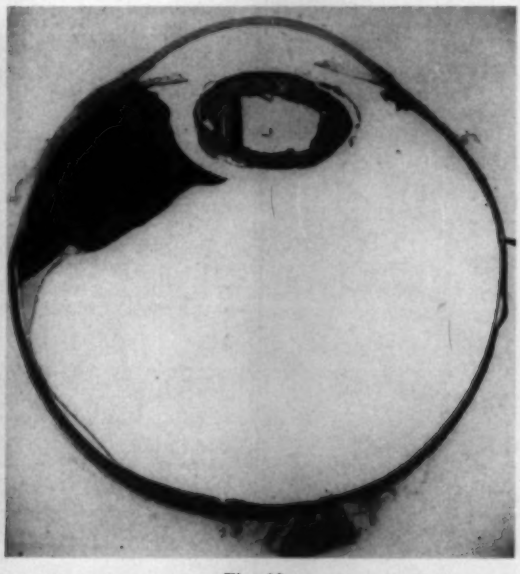


Fig. 10

Figs. 9 and 10 (Baum and Greenwood). (Left, fig. 9) Ultrasonogram of eye shown in Figure 8. (Right, fig. 10) Histopathologic section through approximately the same area.

Fig. 11 (Baum and Greenwood). Fundus photograph of the macular lesion. (Patient of Dr. Max Singer.)

Fig. 12 (Baum and Greenwood). Ultrasonogram of macular lesion shown in Figure 11. Note the area of thinning (arrow). (1) Cornea. (2) Iris. (3) Lens. (4) Macular lession. (5) Posterior pole of the eye and orbital fat. (6) Passage of optic nerve through sclera.

angioma of the choroid proved to be the cause of the macular lesion (fig. 13). Radioisotope studies, utilizing P^{32} uptake were negative.

Vitreous hemorrhage

A hemorrhage into the vitreous may so obscure the fundus to light that, with existing ophthalmic instrumentation, it becomes impossible to determine whether there is an additional associated pathologic process. A differential diagnosis may be made by ultrasonography.

The pattern of the ultrasonogram of a simple vitreous hemorrhage is characterized by weak, diffusely distributed echoes from the vitreous cavity. These will shift and vary with the motion of the eye (figs. 14 and 15).

Radiolucent intraocular foreign bodies

Another major advantage of ultrasonic visualization is its ability to visualize and precisely locate radiolucent foreign bodies (fig. 16). Such localization may be performed through light opaque media. Foreign bodies may readily be distinguished even in the presence of amounts of blood which would render the fundus opaque to light

Fig. 13 (Baum and Greenwood). An angioma of the choroid proved to be the etiology of the macular lesion described in Figures 11 and 12.

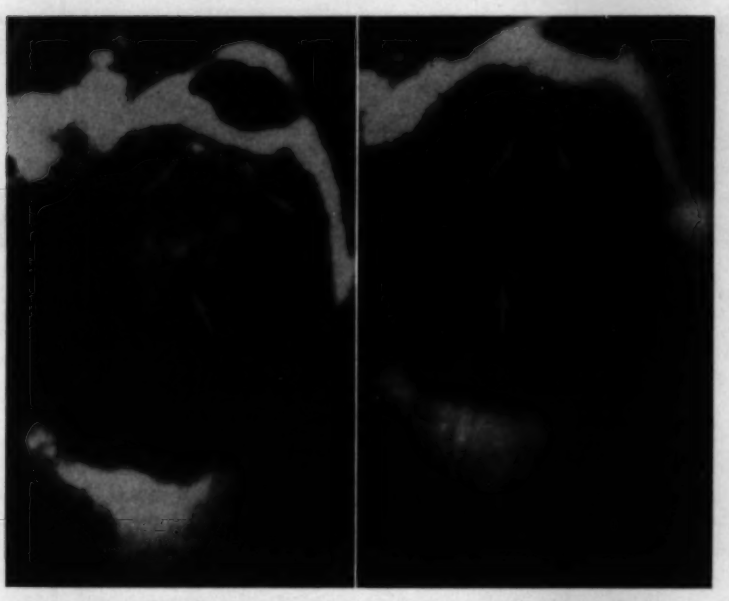


Fig. 14 Fig. 15

Figs. 14 and 15 (Baum and Greenwood). Ultrasonograms of an aphakic eye with a massive vitreous hemorrhage. Note the faintness of the echo and its random distribution.

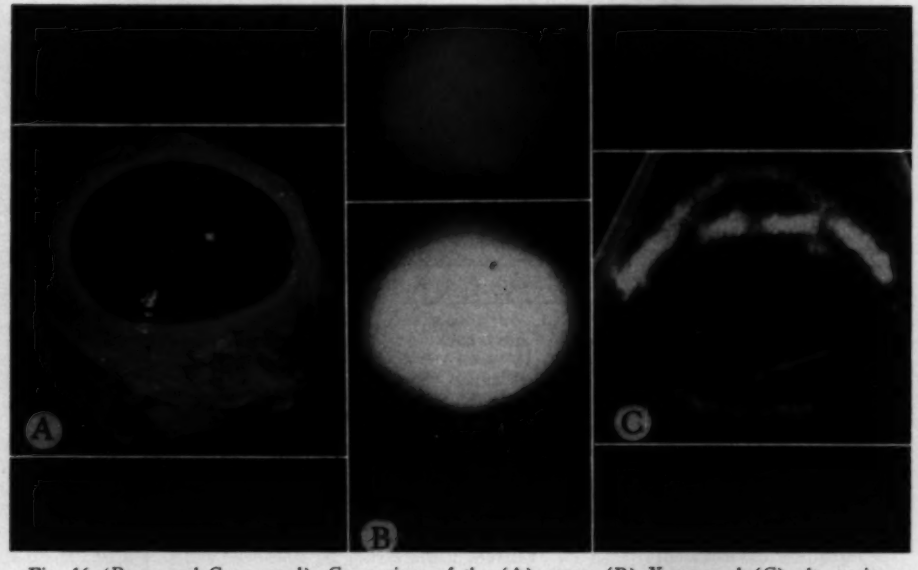


Fig. 16 (Baum and Greenwood). Comparison of the (A) gross, (B) X-ray, and (C) ultrasonic appearance of an eye containing a piece of wood in the vitreous. The arrow on the ultrasonogram points to the sonic image of the wood. It is invisible to X-ray localization. The precise position of the foreign body as to depth and azimuth may be found directly by the ultrasonic locator. (Beef eye specimen.)

Fig. 17 (Baum and Greenwood). The appearance of blood and wood in the vitreous (the eye is viewed laterally through the sclera). (A) Viewed with the full strength transmitted sound beam. (B) Partially attenuated transmitted sound beam. (C) Attenuation of transmitted beam is increased to a point at which the wood remains visible while the blood disappears, thus permitting localization of the wood in the presence of blood in the vitreous. (Beef eye specimen.)

(fig. 17). The same techniques may be applied in the orbit. Radiopaque foreign bodies may also be visualized and localized without the use of external markers, which are ordinarily required for localization by X-ray films.

Table 1 lists the advantages of ultrasonic visualization over X-ray visualization.

Orbital applications

The optic nerve, check ligaments, and muscles have been visualized. Orbital tumors, cysts and foreign bodies have also been demonstrated.

Tumors of the orbit

Heretofore the location of orbital tumors could only be surmised. X-ray examination offered some assistance only if bone erosion occurred. Contrast media proved irritating and occasionally produced severe side-reactions. Radioisotopes could not be used because of the depth of these lesions.

Ultrasonography permits these lesions to be visualized.

A unilateral proptosis of this patient's right eye had been noted (fig. 18). X-ray films demonstrated erosion of the frontal process of the zygoma, but failed to show the soft tissue mass (fig. 19). An ultrasono-

TABLE 1

Advantages of ultrasonic over X-ray visualization of soft tissues

Ultrasonic Visualization of Soft Tissues	X-ray Visualization of Soft Tissues
1. The soft tissues may be visualized directly without the use of contrast media. The detail is equivalent to a low-power photomicrograph	1. Soft tissues have uniform density to X rays. Contrast media are required, but the detail fails to equal that obtained by ultrasonic visualization. Contrast media are irritating and possess toxic side effects
2. Safe at the levels used for visualization. No immediate, cumulative, or delayed effects	2. Danger of X-ray exposure and cumulative effects
3. Depth and angular location can be directly measured without markers	3. Markers required
4. Most forms of foreign materials may be visualized and localized	4. Many substances possess the same X-ray density as the soft tissues in which they are embedded and cannot be visualized
5. Visualization and localization may be performed in any part of the eye or orbit	5. Radioisotope localization and identification of tumors is dependent upon their anterior location and selective uptake

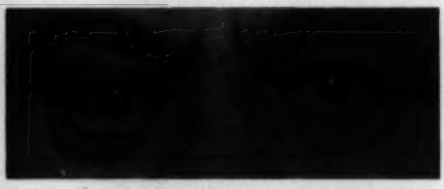


Fig. 18 (Baum and Greenwood). Patient with proptosis of the right eye. Retinal folds were visible in the fundus. These were produced by the pressure of the orbital mass. (Patient of Dr. Benjamin Friedman.)

gram of this patient's eye and orbit demonstrated the orbital tumor (fig. 20). Part of the tumor indented the globe. Retinal folds caused by this pressure could be observed with the ophthalmoscope. An ultrasonogram of the left eye and orbit of this patient did not show any such mass (fig. 21). These findings were confirmed at operation (fig. 22).

We believe this is the first report of the visualization of an intraorbital soft tissue mass prior to surgery.

Orbital foreign bodies

Intraorbital radiolucent foreign bodies can also be visualized by ultrasonography.

A polyethylene tube had been inserted about the sclera of this eye in the equatorial zone for treatment of a retinal detachment. The procedure failed to reattach the retina and the orbit became secondarily infected. Shortly after this ultrasonogram was taken the tube had to be removed. Figure 23 is the ultrasonogram which demonstrated the presence of this foreign material. An ultrasonogram of the normal, unoperated eye of this patient showed no echo suggestive of a foreign body (fig. 24). This, too, is the first time an intraorbital radiolucent foreign body has been demonstrated prior to surgery.

Fig. 19 (Baum and Greenwood). Skull film of patient shown in Figure 18. Note erosion of the frontal process of the zygoma of the right eye (arrow). (Patient, in Figure 18.)

Safety of this type of examination

Both animal experiments and clinical experience indicate that it is safe to expose the eye to the level of ultrasound produced in this equipment. Its average power is less

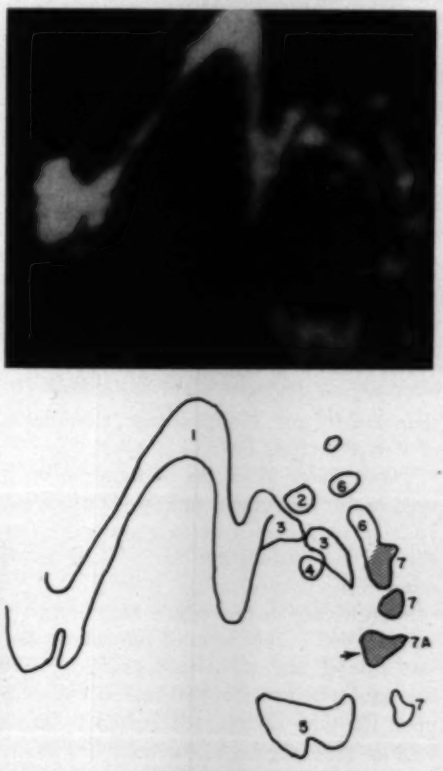


Fig. 20 (Baum and Greenwood). Ultrasonogram of the right eye. Note tumor mass indenting the side of the globe (arrow). (1) Nose. (2) Cornea. (3) Iris. (4) Lens. (5) Posterior pole of the eye and orbital fat. (6) Lid. (7) Orbital tumor. (7A) Portion of tumor indenting the globe and causing retinal folds. (Patient shown in Figure 18.)

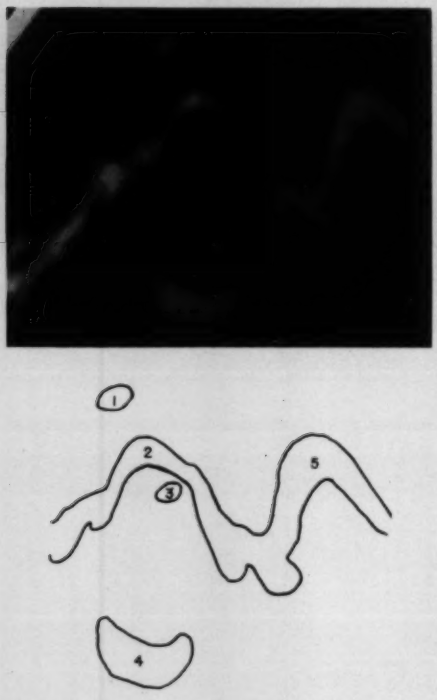


Fig. 21 (Baum and Greenwood). Ultrasonogram of normal left eye. (1) Lid. (2) Iris. (3) Lens. (4) Posterior pole of eye and orbital fat. (5) Nose. (Same patient as in Figures 18, 19, and 20.)

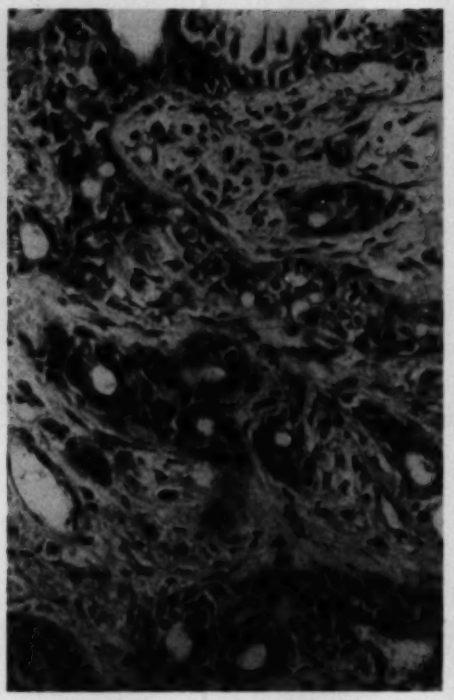


Fig. 22 (Baum and Greenwood). The proptosis of the patient shown in Figure 18 was caused by a mixed tumor of the lacrimal gland. (Same patient as in Figures 18, 19, 20, and 21.)

than 0.07 w/cm.^{2}. No immediate, cumulative, or delayed effects have been noted.

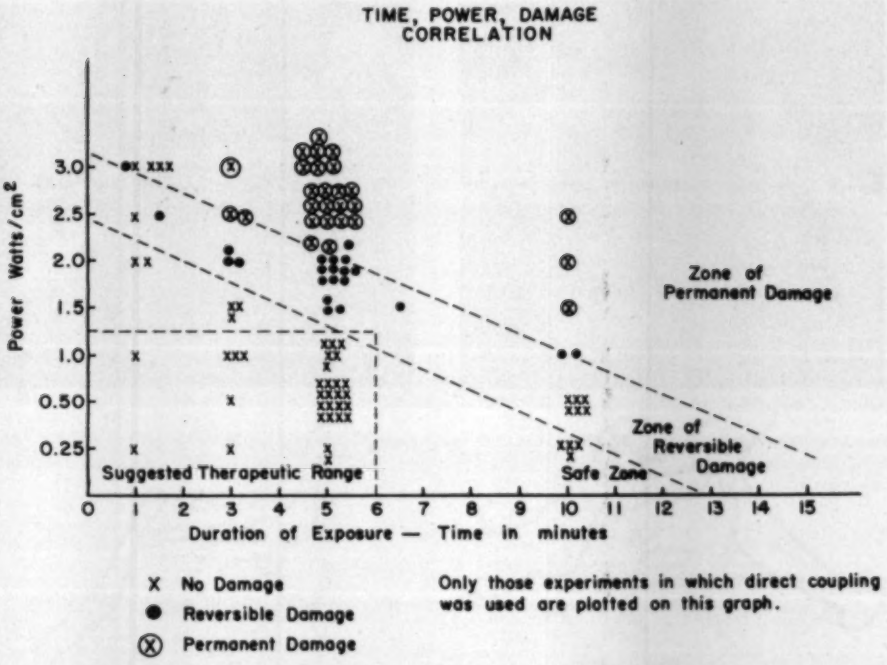
Tissue doses in excess of those normally used in this instrument failed to produce any reaction or lesions in exposed eyes. Exposure of the human eye for as long as 45 minutes has proved safe.

French[2] has demonstrated that brain tissue exposed to this form of ultrasound does not exhibit any pathologic change. Wild[3] has used ultrasound of this intensity directly upon the dura of exposed human brain, as well as other organs of the body, for example, breast, uterus, intestine. In no instance has any evidence of acute or delayed injury been noted. Mundt and Hughes[4] have exposed the human eye to such irradiation without harmful effect. Howry[5] has reported a similar absence of effect following exposure of the breast, neck, extremities and abdomen of human patients.*

Other forms of ultrasound used in ophthalmology

Low power 0-4.0 w/cm.^{2}. Ultrasonic radiation of the eye for exudates and hemorrhages by low intensities of radiation had yielded unsatisfactory therapeutic results.[6-10] Energy levels in excess of 1.0 w/cm.^{2} for three minutes undoubtedly are injurious to the eye[11] (graph 1), and for this reason ophthalmologic guidance should be sought

* Subsequent to the preparation of this manuscript, Oksala, A., and Lehtinen, A., have published data which further attests to the safety of this form of ultrasound applied to the eye.



Graph 1 (Baum and Greenwood). Scattergram of rabbit eyes exposed to *continuous* low-intensity ultrasonic radiation. The center of the transducer was applied to the center of the cornea. Petrolatum was used as the coupling agent. These studies were performed with an unfocused transducer at 1.0 mc.

when ultrasound is to be used about the eye.[12]

High power—approximately 1000 w/cm.² for one to three seconds per exposure. Intense prolonged ultrasound may produce three types of lesions within the eye. These are coagulation of tissue caused by a heating effect, bubble formation secondary to cavitation phenomena, and displacement of cells in the retina when irradiation is performed in the presence of air. Zeiss,[13] Lavine,[14] and Donn[15] have produced such lesions in the cornea and lens. Coagulation produced permanent opacities, while opacities caused by cavitation were reversible. Donn modified the conditions under which radiation was performed by injecting air into the vitreous. Because air is a perfect reflector of ultrasound and also facilitates cavitation he was able to liquefy the vitreous and produce retinal lesions. In the absence of air, he could not succeed in liquefying the vitreous, despite the use of a power level several thousand times greater than that of the ultrasonic locator. Because the introduction of air into the vitreous so disturbs the normal milieu it is difficult to evaluate the significance of Donn's results.

Bell[16] removed the outer of two layers of the frog embryo without affecting the growth potential of the underlying layer. The remaining basal layer formed a completely normal skin, despite the removal of the external layer of cells by ultrasound of 240 w/cm.² at 4.0 mc. He has also produced micro-lesions, that is, lesions affecting only 50 neurons, in the cat brain with focused ultrasound of 2.7 mc. at an intensity of 1700 w/cm.² for 0.25 seconds. Some of the capillaries within such lesions appear to have

Fig. 23 (Baum and Greenwood). Eye demonstrating a persistent retinal detachment and polyethylene tube in situ (arrow). (1) Cornea. (2) Iris. (3) Intraorbital polyethylene tube. (4) Detached retina. (5) Orbital fat. (6) Lateral wall.

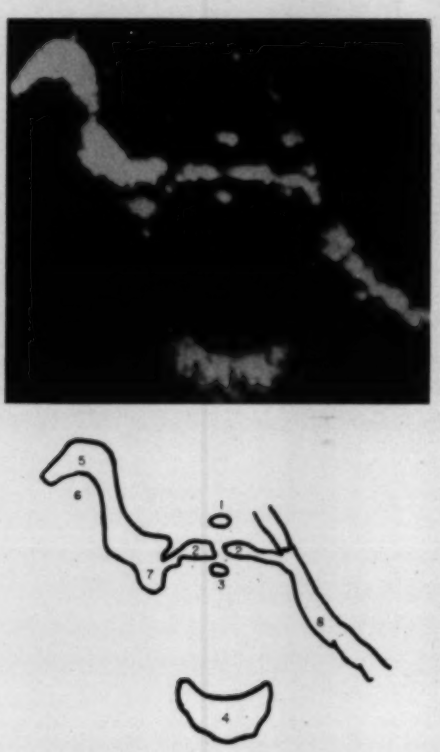


Fig. 24 (Baum and Greenwood). Ultrasonogram of the normal eye of the same patient as demonstrated in Figure 23. (1) Cornea. (2) Iris. (3) Lens. (4) Orbital fat. (5) Nose. (6) Nasal cavity. (7) Nasolacrimal sac. (8) Lateral wall.

been spared, despite necrosis of the surrounding neural elements.

Fry[17] has produced lesions 0.5 to 1.0 mm. in diameter in the central nervous system of the cat without in any way injuring the adjacent neural tissue. When the exposure time was shortened, totally reversible lesions were produced.[18]

Conclusions

Such intraocular pathologic conditions as retinal detachment, tumors, and vitreous hemorrhages have been visualized by ultrasonography in the light-opaque eye. Orbital structures, foreign bodies and pathology have also been demonstrated. Radiolucent foreign bodies have been visualized through the sclera and identified despite blood in the vitreous. Precise localization of such foreign bodies can be made by ultrasound, without the need for external markers employed in X-ray localization.

This paper contains the first report of the ultrasonic visualization of a tumor and radiolucent foreign body within the orbit and is a synopsis of all the forms of ultrasound which have been used in ophthalmology.

Improved instrumentation and further clinical experience hold great promise for future diagnostic applications of the ultrasonic locator.

333 King Street.

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STENOSIS OF THE AQUEDUCT OF SYLVIUS*

L. P. Lassman, F.R.C.S., J. F. Cullen, D.O. Lond., and J. M. L. Howat, D.O. Oxon.

Newcastle upon Tyne, England

This communication is presented to record an unusual defect of the visual fields encountered in a child, aged 10 years, suffering from stenosis of the aqueduct of Sylvius. It is our intention to discuss the abnormalities of the visual fields in cases of this nature and an attempt will be made to explain the field defect found in the case to be described.

In the literature on stenosis of the aqueduct in children, scant attention has been paid to the visual fields and in most cases the fields have either been within normal limits or have not been adequately described. Although stenosis of the aqueduct is a well known clinical entity, in our experience it is of rare occurrence and at this age detailed clinical examination of the visual fields may prove difficult. In this case the child was very co-operative and fairly accurate investigation was possible.

Owing to the small caliber of the aqueduct of Sylvius even slight swelling of its ependymal lining could lead to obstruction and to the production of hydrocephalus. Russell (1949) remarks that whereas the ependyma is highly vulnerable and has little regenerative power, the subependymal glia are readily stimulated to proliferation.

* From the Department of Neurosurgery, Newcastle General Hospital.

Spiller (1902) described the commonest pathologic process involved, and histologic examination of a fatal case in his series showed that the stenosis had been effected by a proliferation of the normal subependymal glia around the aqueduct. This appearance was confirmed by Stookey and Scarf (1936) when they described 16 cases, 12 of which were subjected to adequate histologic examination.

Pennybacker (1940) in a description of 18 cases of non-neoplastic stenosis of the aqueduct states that these subependymal glial cells (and especially their fibrillar elements) seem to proliferate, and may cause a true stricture of the aqueduct from without; or the proliferation may break through the ependyma to form tufts within the lumen which may be valvelike or membranous; or there may be a more or less regular spread of the glial processes within the aqueduct, encroaching on its lumen from within. In yet another variety, the aqueduct may be split into a number of tiny channels which may be almost invisible to the naked eye; the aqueduct becomes a swamp through which the cerebrospinal fluid seeps, but with considerable difficulty.

Russell (1949) mentions that on longitudinal section the anterior part of the aqueduct is funnel-shaped, the stem of a funnel narrowing to a point, which may be anywhere along its course, where the lumen appears to be entirely occluded. This appearance is well visualized in the myodil ventriculogram of the case to be described (fig. 2). Russell (1949) states that microscopically the essential change is an overgrowth of the subependymal neuroglia, which is of a densely fibrillary character and that this may cause displacement, narrowing or even subdivision of the lumen.

The obstruction of the aqueduct gives rise to dilation of the third ventricle and enormous dilation of the lateral ventricles. Rowbotham (1958) is of the opinion that the ventricular dilatation is much greater than is found in cases of posterior fossa tumor. Stenosis of the aqueduct is usually found in plump children with large heads, and it is likely that the obesity may be due to damage to the hypothalamus and that the large head occurs because the internal hydrocephalus develops while the skull is still soft enough to stretch and the sutures to give way. The cases of this syndrome often present with headaches and deterioration of vision and the differential diagnosis from suprasellar tumor, posterior fossa tumor, midbrain tumor or pinealoma may be difficult, but with the introduction of myodil ventriculography the diagnosis may be confirmed with certainty. Air ventriculography may be dangerous in cases of pineal tumor and, if this is the most likely diagnosis, myodil should be used in preference to air.

If a visual field defect is found in a case of this nature, it is presumably due to a dilated third ventricle pressing on the upper surface of the chiasma. This occurs in the midline region and involves the crossed fibers from the nasal halves of the retinas producing a bitemporal hemianopsia without special characteristics. In such circumstances one would naturally expect the uppermost fibers in the chiasma to be involved first, as in any suprasellar tumor, and this would give rise to inferior quadrantic field defects as described by many observers—Duke-Elder (1949), Hughes (1954) and Scott (1957).

Other field defects have been mentioned by various authors in cases of dilatation of the third ventricle. Binasal defects are suggested by Duke-Elder (1949) to be due to crowding of the optic nerves against the resistant vessels of the anterior part of the circle of Willis. Walsh (1957) suggests that in such cases the optic nerves are pushed laterally against the internal carotid arteries. Hughes (1954), however, is of the opinion that the incrimination of the anterior cerebral arteries is false, as he has found them to be actually lifted away from the chiasma and optic nerves. Both Hughes (1954) and Duke-Elder (1949) mention paracentral scotomas as occurring with relative fre-

quency in these cases, due to early involvement of the posteriorly situated macular fibers. Pennybacker (1940) describes a case in which there were bilateral central scotomas due, in his opinion, to compression of the optic nerves by the dilated third ventricle, and Hughes (1954) again states that small paracentral scotomas in the apex of the inferior temporal quadrant are the first changes to occur—these are usually bilateral, but are sometimes more advanced on one side than the other.

Wagener and Cusick (1937) describe other field defects in cases of dilatation of the third ventricle:

1. Central scotomas in each eye.
2. Unilateral central scotoma with temporal hemianopsia in the other eye.
3. Unilateral amaurosis and temporal hemianopsia in the other eye.
4. Complete homonymous hemianopsia.
5. Bilateral blindness.
6. Incomplete bitemporal defects.

They were dealing, however, with cases where the dilated ventricle was secondary to posterior fossa tumors. Homonymous hemianopsia is mentioned by Jefferson (1945) as occurring in cases of dilatation of the third ventricle, and Duke-Elder (1949) also mentions this defect, which in his opinion is due to compression of the optic tracts by the posterior communicating arteries.

In the case to be described the field defect was an upper quadrantic temporal loss in the left eye. This is what one would rather expect from a lesion situated below the chiasma or optic nerve, indicating as it does pressure on the inferior surface of one or other of these structures. From all other aspects this was a case of stenosis of the aqueduct with third ventricular dilatation.

Case history

A boy, aged 10 years, was first seen in late January, 1958, because of severe headaches and slight personality changes. Six weeks previously he had developed a "flulike" illness with fever, malaise, headaches, dizziness, vomiting and anorexia, and there had been marked deterioration in his work at school.

Examination revealed no abnormal physical signs; visual acuity was right 6/5, left 6/6; the optic discs were flat and the visual fields were plotted and were found to be well within normal limits.

X-ray films of the skull showed that the pituitary fossa was normal in size; the convolutional markings on the inner table appeared to be accentuated

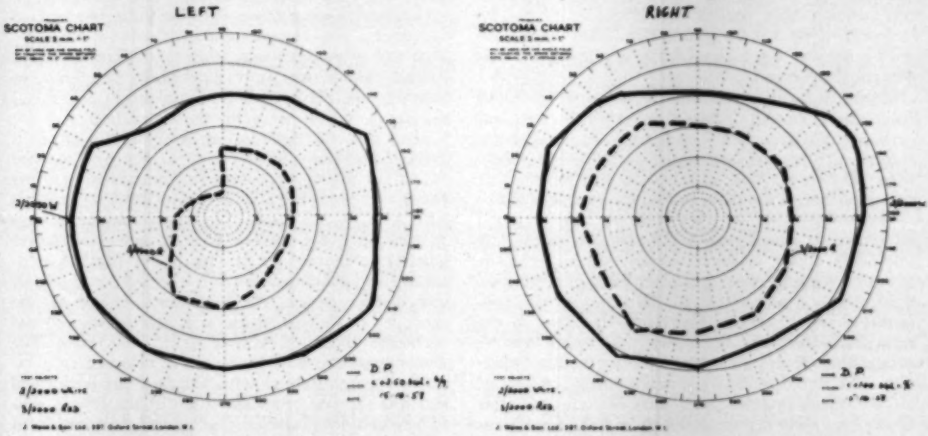

Fig. 1 (Lassman, Cullen and Howat). The preoperative visual fields. There is a well-marked upper temporal quadrantic defect in the left eye to 3/2,000 red, and a less well-marked similar defect to 2/2,000 white. (Note: In Figures 1 and 3 the blindspots were not grossly enlarged and were unconnected with the field defect. The emphasis in plotting these fields was laid on the isopters rather than on the blindspots. For clarity of presentation the blindspots are not shown in the diagrams.)

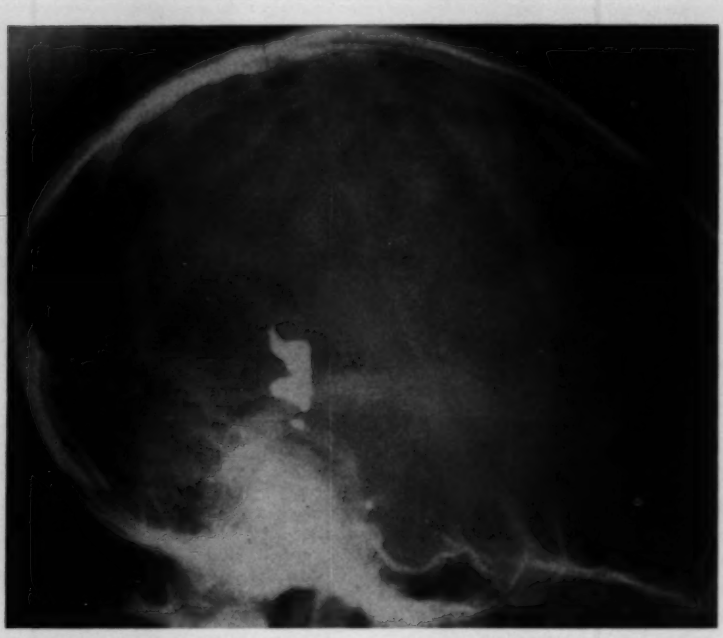

Fig. 2 (Lassman, Cullen and Howat). Myodil ventriculogram. Myodil is shown in the upper end of the aqueduct which does not appear to be displaced. No myodil passed through the aqueduct, and at a later examination after 24 hours had elapsed, the myodil in the upper end of the aqueduct remained in the same position. This appearance was characteristic of aqueduct stenosis.

but there was no evidence of any definite suture diastasis in the sagittal suture. The electro-encephalogram was normal. A diagnosis of benign hydrocephalus, which could have been associated with a sagittal thrombophlebitis, was made and observation arranged.

At the follow-up examination in April the right optic disc showed slight but distinct temporal pallor, which was confirmed in June. Otherwise the clinical picture was unchanged on both these occasions. The visual field examination was repeated at each visit and again no abnormality was detected.

On July 21st he had a generalized convulsion and on return to consciousness the reflexes on the left side were found to be increased and an extensor plantar response was present. The cerebrospinal fluid pressure was raised to 280 mm. but the constituents were normal. The temporal pallor in the right optic disc was still present but there was no suggestion of any incipient papilledema in either disc. The visual fields were again normal. A second electro-encephalogram was carried out and this showed no abnormal features. A right percutaneous carotid angiogram was then performed and this suggested dilatation of the ventricles. At this stage, however, as the child was so well no further investigations were advised and observation was again arranged.

The boy was seen again in September and as the optic discs now showed a definite early papilledema, arrangements were made for him to be re-admitted to hospital. On examination in hospital he had no abnormal neurologic sign other than bilateral papilledema, which was more marked in the right eye than the left. He had had a recurrence of occasional headaches, which were of short duration and not violent, and he had put on weight. Visual field examination now showed a slight but well marked "slant" in the upper temporal quadrant of the left eye to the two mm. white test object. Further analysis with a target of lesser intensity (three mm. red) showed that the defect had definite hemianopic quadrantic characteristics (fig. 1). A third electro-encephalographic examination was not diagnostic of any definite lesion. Straight X-ray films of the skull now showed definite suture diastasis and markedly increased convolutional markings. The diameter of the skull measured 22.5 inches.

On October 16th a ventriculogram was performed and this showed gross dilatation of the lateral ventricles and the anterior portion of the third ventricle, but air would not pass into the posterior part of the fourth ventricle.

On the straight films the volume of the posterior fossa did not appear to be significantly increased, although the remainder of the head was extremely

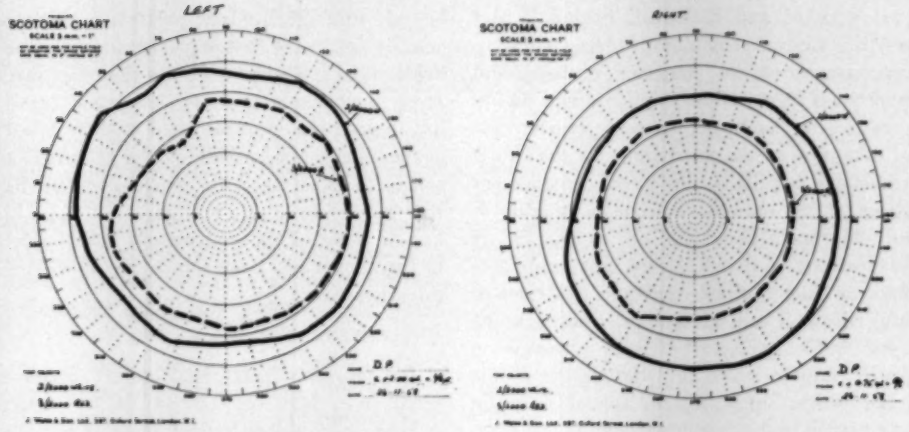

Fig. 3 (Lassman, Cullen and Howat). The postoperative visual fields. This shows that the upper temporal quadrantic defect to 2/2,000 white has resolved, and that to 3/2,000 red is much less marked.

large. The diagnosis of aqueduct stenosis was made and a Torkildsen's operation performed, using a rubber catheter from the left lateral ventricle to the cisterna magna.

After this operation his condition began to settle satisfactorily and on October 31st a myodil ventriculogram was performed, using 0.75 cc. of myodil in an attempt to confirm the diagnosis. The appearances were consistent with aqueduct stenosis (Fig. 2).

Following operation the papilledema gradually subsided and on November 5th the visual fields were plotted and showed an intermediate stage bebetween Figures 1 and 3. When the visual fields were again plotted on November 26th, there was a definite regression of the visual defect (Fig. 3). Since then the boy has been perfectly well and has returned to his normal activities.

Comment

An upper temporal quadrantic field defect is commonly found in the early stages of pituitary tumors, and the field changes are produced by pressure on the inferior surface of the chiasma in the midline. Visual field defects due to such tumors are best demonstrated, in the early stages, on the Bjerrum screen with small test objects such as the two mm. white, or three mm. red targets, and may be evident in only one eye. In the case described further analysis of the defect, with its hemianopic characteristics, demonstrated that we were not dealing with a simple depression of the field due to developing optic atrophy, but that there was some direct involvement of the nerve or chiasma. The sella was normal in size and there was no evidence which would point to an expanding intrasellar lesion. A lesion at the chiasmal end of the left optic nerve could produce the field defect described, and here again the pressure would have to be on the lower surface of the nerve, but no evidence has been produced of any lesion which could cause direct pressure in this area.

There is no doubt that the cause of the field change must be ascribed to the enormously dilated third ventricle demonstrated on the ventriculogram. This is confirmed by the fact that the defect was found to have regressed when the visual fields were plotted again (fig. 3), six weeks after the Torkildsen's operation. It is interesting to speculate why the dilated third ventricle has not caused damage to the upper surface of the chiasma and produced the customary inferior quadantic field changes as described by other observers.

In its normal anatomic position the third ventricle lies above and behind the optic chiasma, even when the latter is postfixed in position. In the case of pituitary tumors a prefixed chiasma will allow a swelling to

pass upward and backward behind it and produce central scotomatous changes due to involvement of its posterior surface, and sometimes inferior quadrantic defects due to pressure on its upper surface. In the case of dilatation of the third ventricle such variations are less likely, due to the constant position of the normal ventricle in relation to the chiasma, irrespective of its fixation. It has been stated that when the chiasma is prefixed a dilated third ventricle can burrow underneath it and even appear between the optic nerves anteriorly. Such a condition could cause a bitemporal depression in the upper fields, but hardly an isolated lesion in one eye, as in the case described.

The most likely explanation is that the enlarged third ventricle, forming as it were a soft swelling above the chiasma has pushed this structure down against a more resistant surface below, thus causing damage to its under aspect. It is also probable that the chiasma has been pushed forward as well as downward due to the anatomic relationship between the chiasma and ventricle described above. This movement of the chiasma and proximal ends of the optic nerves would cause them to impinge against the upper surface of the tuberculum sellae, or its lateral aspect. In this way the damage would be caused to their under surface and an upper quandrantic field defect as described would be produced. In the case of the chiasma this defect could be an early one and still unilateral, although in these particular circumstances one would have expected to find some evidence of a similar defect in the other eye. It is more likely that the left optic nerve has been pressed downward and forward against the lateral aspect of the tuberculum sellae. In either case the conclusion must be that the lesion is due to indirect damage to one or other of these structures rather than to direct damage from the overlying dilated third ventricle.

Summary

An unusual visual field defect encountered in a case of stenosis of the aqueduct of Sylvius is described. A short account of the likely pathology of the lesion is given, and a note made of the dilatation of the ventricular system and especially of the third ventricle. A brief review of the literature of the field defects accompanying third ventricular dilatation is given and an attempt has been made to explain the cause of the field defect in the case described.

Westgate Road (4).

Acknowledgements

We wish to thank Dr. S. Woolf for referring the patient to our department and for his interest in the case; Dr. Gordon Gryspeerdt, neuro-radiologist, and Dr. Christine Cooper, consultant pediatrician, Newcastle General Hospital, for their advice and assistance with the treatment of the child. Our grateful thanks are also due to Prof. Brodie Hughes for his very helpful advice in the preparation of this paper.

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CEPHALOSPORIUM KERATITIS*

J. L. Byers,† M.D., M. G. Holland,† M.D., and J. H. Allen, M.D.
New Orleans, Louisiana

Since 1839 when Langenbecher[1] first discovered fungi in human tissue, there have been many reports of fungus infections in man. However the use of broad-spectrum antibiotics and of steroid preparations has contributed greatly to the increased incidence of mycotic infections. Sometimes nonpathogenic fungi have been made pathogenic by use of these agents.[2] Indeed, the laboratory animal often must be pretreated with steroids in order to facilitate growth of the fungus in question.[3] At times fungus infections complicate other diseases, such as leukemia, Hodgkin's disease, tuberculosis, and diabetes, in which the patient's general resistance to infection has been lowered.[4]

The purpose of this paper is to report two cases of corneal ulceration from which a cephalosporium species of fungus was cultured. Both patients had received antibiotics and very probably steroids before fungus cultures became positive. Only three other cases of ocular involvement by this fungus have been reported. All of them involved the cornea: Serra[5] in 1929, Bedell[6] in 1946, and Ley[3] in 1956.

The cephalosporium fungus is related to Tricophyton gypseum,[7] and in 1955 was found by Sir Howard W. Florey[8] to produce several nontoxic antibiotics which showed therapeutic value against diphtheria and tetanus bacilli.

Bedell,[6] in 1946, cultured a cephalosporium species of fungus from a corneal ulcer in a farmer, who had received an abrasion from a cow's tail. A cure was effected with potassium iodide therapy after preliminary trial with sulfathiazole and penicillin had failed. Bedell was unable to reproduce corneal ulcers in rabbits with this fungus. Ley, also, was unable to produce keratitis in rabbits with a cephalosporium species even after cortisone treatment.

Case reports

Case 1

W. H., a 47-year-old Negro, was referred to the Tulane Eye Clinic of Charity Hospital by his family physician two weeks after apparently spontaneous onset of pain, photophobia, and foreign body sensation in the left eye. He had been treated unsuccessfully before referral with a variety of topical ointments and solutions containing steroids and antibiotics.

On the admission examination there was intense injection of the left eye with a deep corneal ulcer three mm. in diameter in the inferior paracentral area. There were many large keratic precipitates and a dense flare. Corneal sensitivity was decreased. A small black filamentous "foreign body" was removed from the center of the ulcer. No microorganisms were present on smear or cultures from the base of the ulcer. The patient was admitted to the hospital and treated with scopolamine and sulfacetamide topically and systemic typhoid fever therapy. During the course of fever therapy, he received large amounts of systemic streptomycin. For a few days after therapy was instituted, there was symptomatic improvement; however, hypopyon developed with superficial extension of the ulcer. Since repeated smears and cultures from the ulcer had failed to reveal micro-organisms, an aqueous sample was taken by paracentesis of the anterior chamber but showed no growth on cultures for fungi, viruses and bacteria. Following paracentesis, there was improvement for several days, but healing was incomplete.

The patient left the hospital at this time. He returned two weeks later with a dense paracentral purplish-black ulcer surrounded by a grayish white zone of infiltration. Hypopyon was present (fig. 1).

Scrapings from the ulcer grew cephalosporium fungus on Sabouraud's and Lipman's mediums. The ulcer was cauterized with iodine and oral sodium iodide therapy was started (dose: 1.0 gr. per 10 lb. body weight, tid). After two weeks without response, topical copper-sulfate therapy was added. One drop of 0.125-percent copper-sulfate solution was instilled onto the cornea every hour during the day. Response was slow, with gradual healing of the ulcer in the next eight weeks, at which time the oral sodium iodide and the topical copper sulfate were discontinued. Intermittent episodes of elevated intraocular pressure were controlled with Diamox.

* From the Department of Ophthalmology, Tulane University School of Medicine and Charity Hospital of New Orleans.

† Special trainees, National Institutes of Health, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

We are indebted to Dr. Earl Fisher, Jr., for his work in the laboratory with the cultured material.

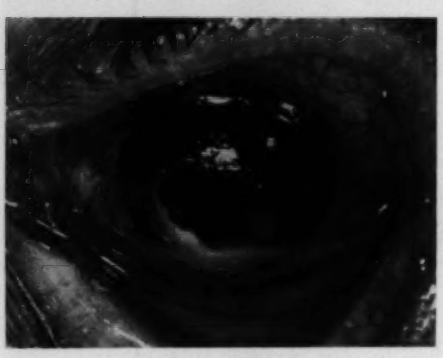

Fig. 1 (Byers, Holland and Allen). Large purplish-black central corneal surrounded by grayish zone of infiltration accompanied by hypopyon in Case 1.

Seven months after the patient was first seen, the corneal ulcer was healed and the eye quiet but the cornea was heavily vascularized between the leukoma and the inferior limbus. An inferior synechialysis and iridectomy were made at six o'clock because of dense adhesions. Six weeks later a seven-mm. circular full thickness keratoplasty was made. The postoperative course was uneventful. Histologic examination of the excised corneal button with special stains revealed scarring with some vascularization but no evidence of fungi.

CASE 2

J. L., a 59-year-old Negro, was referred to the Tulane Eye Clinic by his family physician three weeks after being struck in the right eye by a corn stalk. He had been treated before referral with steroid suspensions topically.

On the admission examination, there was minimal edema of the lids, intense injection of the conjunctiva, a large yellowish-white central corneal ulcer, and hypopyon (fig. 2).

Repeated microscopic examinations and cultures of scrapings from the base of the ulcer yielded no organisms. The patient was admitted to the hospital for treatment with intravenous typhoid H antigen, systemic iodides, and Chloromycetin. After no improvement, he was discharged three weeks later on topical scopolamine, Chloromycetin, and oral sodium iodide.

At the time of the second admission, two weeks later, a cephalosporium species of fungus was cultured from corneal scrapings. He was continued on oral sodium iodide and topical scopolamine therapy. Topical 0.125-percent copper sulfate solution and daily copper-sulfate iontophoresis were started. The iontophoresis treatments were stopped after five treatments because of chemical debridement of the ulcer bed, with pinpoint perforation of the ulcer. Following perforation of the ulcer, there was noticeable improvement and healing.

Four months later, an eight-mm. partial penetrating keratoplasty was performed. The postoperative course was uneventful. Examination of the excised corneal button revealed scarring and vascularization but no fungi were present.

DISCUSSION

As laboratory methods have improved for the diagnosis of fungus infections, an increasing incidence of ocular fungus infections has been observed. Ley and Sanders[2] have emphasized that there has been an increased incidence of keratomycosis since the introduction of antibiotics and steroids, and suggest a causal relationship. Indeed, in order to infect experimental animals with fungi, it was necessary for Ley[3] to pretreat the animals with steroids. It is noteworthy that most cases of keratomycosis diagnosed clinically (not a common event) or histopathologically, have been treated with corticosteroids before the diagnosis was made.

The cases presented in this report are of interest not only because of their infection with a fungus not commonly encountered in keratomycosis but also because of the characteristic history, clinical picture, and the noteworthy response of the lesions to systemic iodide and topical copper sulfate therapy.

The clinical picture of a recalcitrant cor-

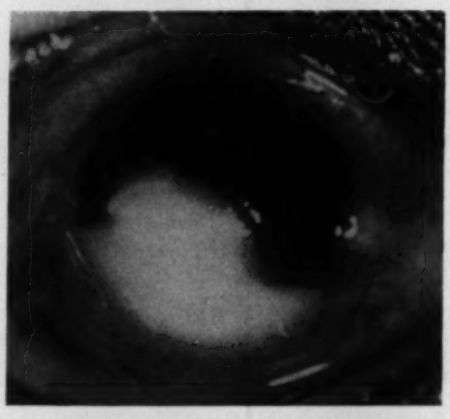

Fig. 2 (Byers, Holland and Allen). Large yellowish-white central corneal ulcer and hypopyon in Case 2.

neal ulcer with hypopyon, which is intractable indeed, flourishes with antibiotic and corticosteroid therapy should immediately suggest keratomycosis, especially if the ulcer shows pigmentation. In the first case presented in this report, the ulcer became deeply pigmented to a purplish-black color. Cultures of cephalosporium obtained from the ulcer did not exhibit this pigment, however.

It apparently is not unusual for initial cultures to fail to reveal the offending organism.

As treatment of these two patients continued without noticeable benefit, one of the authors (J. H. A.) suggested the use topically of 0.125-percent copper-sulfate solution. Copper sulfate has been used as a fungicidal agent in industry and, of course, has been used both in stick form and as drops in the treatment of trachoma. The in vitro sensitivity studies of both cultures of cephalosporium obtained from the reported cases showed an inhibition of growth in dilute 0.06-percent copper-sulfate solution. It is our opinion that the addition of copper sulfate topically materially benefited the healing of the corneal ulcers, although Bedell[8] has reported success with iodides alone in the treatment of this type of keratomycosis.

Recently Mangiaracine and Liebman[9] reported successful treatment of a case of hypopyon keratitis caused by Aspergillus fumigatus with Nystatin. Montana and Sery[10] demonstrated definite inhibition of Candida albicans infections of rabbit corneas by Nystatin and amphotericin B. Sensitivity tests with these agents demonstrated fungistatic activity in fairly low dilutions, but unfortunately the results were not available in time for clinical evaluation in the reported cases. The in vitro sensitivity of cephalosporium to both amphotericin B and nystatin encourages speculation that they will be of benefit in the treatment of this type of corneal infection. Further laboratory studies in this line are now in progress.

Summary

Two cases of hypopyon corneal ulcer, from each of which a species of cephalosporium fungus was cultured, are reported, making a total of five such infections of the cornea recorded in the literature.

In the first case, the cornea exhibited a purplish-black pigmentation which was not present in the culture.

The topical application of 0.125-percent copper-sulfate appeared to influence favorably the healing of the corneal ulcers while the patients were given sodium iodide systemically. The offending organism was definitely inhibited in vitro by dilute solutions of copper sulfate.

The in vitro sensitivity of the cephalosporium fungi isolated from the reported cases to amphotericin B and nystatin suggests that these agents will be of therapeutic value.

1430 Tulane Avenue (12).

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THE EFFECT OF BC-48 (DEMECARIUM BROMIDE) ON NORMAL RABBIT AND HUMAN EYES*

NARENDRA KRISHNA,† M.B.
New Delhi, India
AND
IRVING H. LEOPOLD, M.D.
Philadelphia, Pennsylvania

INTRODUCTION

Recently a new synthetic, powerful, long-acting cholinesterase inhibitor BC-48 (generic name: demecarium bromide; chemical name: decamethylene bis-[N-methyl-carbaminoyl-m-trimethylammonium phenol]), a quaternary ammonium compound, became available. Because of its ready solubility in water and its stability for indefinite periods, it promised to be a useful anticholinesterase agent in control of glaucoma. Consequently the investigations reported here were undertaken to determine the effect of BC-48 on normal rabbit and human eyes. After an initial study, which showed that BC-48 did not cause any permanent damage to the rabbit eyes, studies on the normal human eyes were undertaken.

PART I

EFFECT OF BC-48 ON NORMAL RABBIT EYES

Materials and methods. Young albino rabbits of approximately equal weights were used. Three strengths 0.1, 0.25 and 0.5 percent of BC-48 in aqueous solution were employed. Exact amounts of solution were measured by means of a 1.0 cc. microsyringe and delivered directly to the eyes either in the form of drops or subconjunctival injections. Care was taken to keep the eyes closed by drawing the two eyelids together, for at least one minute after each instillation to attain maximum effect.

Results. MIOSIS. Table 1 shows the effect of BC-48 on miosis. The rapidity of onset of maximal miosis and the duration of sluggish response of the pupil to light were found to be directly proportional to the concentration of BC-48 employed. No cumulative effects were noted when 0.05 cc. of 0.1 percent of BC-48 was instilled into the eye every other day for eight days. Pupillary signs recovered in 24 hours in these eyes just as in other eyes receiving one single instillation.

CONGESTION. The degree of congestion of the eye was found to be directly proportional to the concentration of BC-48 employed: 0.1 percent produced only a mild conjunctival hyperemia lasting a few hours; 0.25 percent produced moderate degree of conjunctival injection and a mild ciliary flush, lasting for 24 hours; 0.5 percent caused marked superficial and deep congestion which lasted for 72 hours.

INTRAOCULAR PRESSURE. Intraocular pressures were measured by means of a Schiøtz tonometer under deep nembutal anesthesia in 18 rabbits. Table 2 shows one set of experiments. Essentially similar results were obtained in the other two sets. Tension-lowering effect of BC-48 lasted more than one week: 0.1 and 0.25-percent solutions lowered the tension by 4.0 mm. Hg or more, 15 minutes after instillation; 0.5-percent solution, however, caused a transient rise in tension by a 3.0 mm. Hg or more in most of the eyes 15 minutes after instillation; this rise

* From the Department of Ophthalmology, Graduate School of Medicine of the University of Pennsylvania, and the Research and Clinical Departments of the Wills Eye Hospital. Presented in part at the Eastern Section meeting of the Association for Research in Ophthalmology, New York University-Bellevue Medical Center, New York, November 21, 1958. BC-48 used in this study was provided by Merck, Sharp & Dohme, Research Laboratories, Division of Merck & Co., Inc., West Point, Pennsylvania, through the courtesy of Dr. Edmund W. J. DeMaar. Trade name: Humorsol.

† Fight for Sight research fellow of the National Council to Combat Blindness, Inc., New York.

TABLE 1

EFFECT OF BC-48 (DEMECARIUM BROMIDE) ON PUPILLARY REACTIONS OF RABBIT EYES

No. of Eyes	0.05 cc. %	Average Appearance of Maximal Miosis Minutes after Instillation	Pupillary Reactions (days)		
			1	2	3
12	0.1	32	Normal	Normal	Normal
12	0.25	24	Sluggish	Normal	Normal
12	0.5	16	Sluggish	Sluggish	Normal

was noticeable even 30 minutes after instillation, but after one hour there was definite lowering of tension.

Lowest tension was recorded with 0.1 percent between one and six hours, with 0.25 percent between 30 minutes and six hours, and with 0.5 percent between six hours and seven days after instillation.

At the end of seven days, tensions were still much below their original values, but maximum recovery was noticeable in eyes in which 0.1 percent solution was used and least recovery in eyes in which 0.5 percent solution was employed. The tensions returned to their original values between 10 to 12 days in all the eyes.

SIDE-EFFECTS. The severity of systemic and local effects was directly proportional to the concentration of BC-48 employed. Similarly there was a more rapid onset of these signs, with higher concentrations. The instillation of BC-48 drops into the conjunctival cul-de-sac was followed by profuse sweating and excessive lacrimation and later on rapid pupillary constriction. Occasionally diarrhea was noted. There was marked weakness and paralysis of the hind legs accompanied by muscle tremors or twitchings and even generalized convulsions. Respiration became deep and labored and death usually ensued from respiratory failure or generalized exhaustion.

Biomicroscopic examinations revealed conjunctival and circumcorneal injection with transient aqueous flare in some of the eyes treated with BC-48. Histologic examination of 12 eyes enucleated at various intervals after instillation of 0.05 cc. of 0.5 percent BC-48, showed no gross or microscopic changes except engorgement of blood

TABLE 2

EFFECT OF BC-48 (DEMECARIUM BROMIDE) ON TENSION OF RABBIT EYES

Rabbit No.	Eye	0.05 cc. %	Before Instillation	(Tension in mm.Hg [Schiøtz] After Instillation							
				15 min.	30 min.	1 hr.	3 hr.	6 hr.	24 hr.	4 da.	7 da.
11	O.D.	0.1	14.72	8.92	8.92	6.24	6.24	5.18	7.48	7.48	10.59
	O.S.	0.25	12.51	8.92	5.74	7.48	6.24	5.18	5.18	5.18	6.24
12	O.D.	0.1	14.72	10.59	10.59	6.24	6.24	7.48	8.92	7.48	10.59
	O.S.	0.5	14.72	17.26	14.72	14.72	8.92	5.18	5.18	5.18	5.18
13	O.D.	0.25	12.51	7.48	5.18	6.24	5.18	5.18	5.18	7.48	8.92
	O.S.	0.1	10.59	6.24	5.74	5.18	5.18	5.18	5.18	7.48	10.59
14	O.D.	0.25	23.51	17.26	7.48	8.92	8.92	7.48	7.48	14.72	17.26
	O.S.	0.5	17.26	20.17	23.51	17.26	10.59	5.18	5.18	12.51	12.51
15	O.D.	0.5	14.72	12.51	12.51	7.48	7.48	7.48	6.24	6.24	5.18
	O.S.	0.1	12.51	8.92	6.30	6.24	6.24	5.18	5.18	6.24	10.59
16	O.D.	0.5	12.51	12.51	12.51	8.92	7.48	6.24	7.48	5.18	5.18
	O.S.	0.25	12.51	8.92	5.18	7.48	7.48	5.18	5.18	5.18	6.24

TABLE 3

TIME OF DEATH OF RABBITS AFTER 0.1 CC./LB. BODY WEIGHT SUBCONJUNCTIVAL INJECTIONS OF CHOLINESTERASE INHIBITORS

Cholinesterase inhibitor %	Rabbit 1 (min.)	Rabbit 2 (min.)	Rabbit 3 (min.)	Rabbit 4 (min.)	Rabbit 5 (min.)	Rabbit 6 (min.)	Average Time (min.)
BC-48 0.5	7	5	6	6	5	7	6
217-MI 0.5	5.5	7	5.5	7	5	6	6
DFP 0.1	Living after 30	Living after 30	Living after 30	Living after 30	Living after 30	Living after 30	Living after 30
Neostigmine 5.0	4	3.5	4.5	3	4.5	4.5	4
Physostigmine 1.0	12	10	11	11	10	12	11

vessels of conjunctiva, iris and ciliary body.

An attempt was made to determine the time interval during which the death of rabbits ensued from the moment the various commonly used anticholinesterase agents in the recommended maxim therapeutic dosage were given in the form of drops or subconjunctival injections. Tables 3 and 4 show the results.

Among the long-acting cholinesterase inhibitors the average time interval during which death ensued was six minutes both for BC-48 and 217-MI, when injected subconjunctivally, but was shortest with BC-48 (average time two hr.) than with 217-MI (average time 18 hr.) and DFP (average time more than 24 hr.) when given in the form of drops.

SYNERGISMS AND ANTAGONISMS. An attempt was made to determine the toxic pharmacologic relationship between BC-48 and other cholinesterase inhibitors. Table 5 shows the results of one set of experiments. Essentially similar results were obtained in other two sets. Physostigmine when given first blocked the action of subsequent subconjunctival injection of DFP and 217-MI (average time of death 105 minutes and 34 minutes respectively as compared to 4.5 minutes and 4.25 minutes respectively when the order of injections was reversed). No such blocking mechanism was demonstrated in the case of BC-48 by physostigmine (3.5 minutes compared to 4.5 minutes).

PART II

EFFECT OF BC-48 ON NORMAL HUMAN EYES

Materials and methods. Human volunteers, irrespective of age, sex and color were used. The ages ranged from 18 to 70 years. All these subjects were examined in the clinic of Wills Eye Hospital and were included in the present study after all ocular pathology had been excluded apart from refractive error.

BC-48 in the form of neutral aqueous solution in the concentrations of 0.1, 0.25 and 0.5 percent was employed; 0.1 cc. was instilled on the cornea by means of a 1.0 cc. microsyringe while the patient lay horizontal. The lids were held apart for 10 seconds, to allow the cornea and conjunctiva to be bathed. Afterward the lids were held to-

TABLE 4

TIME OF DEATH OF RABBITS AFTER 0.2 CC./LB. BODY WEIGHT DROPS INSTILLATION OF LONG ACTING CHOLINESTERASE INHIBITORS INTO THE EYES

Cholinesterase inhibitor %	Rabbit 1 (hr.)	Rabbit 2 (hr.)	Rabbit 3 (hr.)	Rabbit 4 (hr.)	Rabbit 5 (hr.)	Rabbit 6 (hr.)	Average Time (hr.)
BC-48 0.25	1.5	2	2.5	1.5	1.5	3	2
217-MI 0.25	18	16	20	19	19	16	18
DFP 0.1	Living after 24	Living after 24	Living after 24	Living after 24	Living after 24	Living after 24	Living after 24

TABLE 5

TOXIC PHARMACOLOGIC RELATIONSHIP BETWEEN CHOLINESTERASE INHIBITORS IN RABBITS

Cholinesterase Inhibitor	%			No. of Rabbits	Average Time of Death After 2nd Injection (min.)
1st Injection*		2nd Injection†			
BC-48	0.5	Physo.	1.0	6	3.5
Physo.	1.0	BC-48	0.5	6	4.5
217-MI	0.5	Physo.	1.0	6	4.5
Physo.	1.0	217-MI	0.5	6	34
DFP	0.1	Physo.	1.0	6	4.5
Physo.	1.0	DFP	0.1	6	105

* 0.05 cc./lb. body weight subconjunctival injection.
† Five minutes after 1st injection 0.05 cc./lb. body weight subconjunctival injection.

gether, closing the palpebral fissure for at least two minutes.

Atropine sulfate (one-percent solution) and homatropine hydrobromide (four-percent solution) were employed to induce cycloplegia and to evaluate the counter effects on BC-48. Visual acuities were recorded with Snellen's charts. Intraocular pressures were measured with Schiøtz tonometer. Coefficients of aqueous outflow facilities were determined with Mueller electronic tonometer connected to a Leeds and Northrup recorder.

Results. Single instillations of BC-48 produced miosis in all 40 normal eyes of 20 subjects. Miosis became noticeable in all eyes within 45 to 60 minutes. Maximum miosis was achieved between two to four hours, the pupils becoming pinpoint. Miosis persisted on the average between three to 10 days. But in two cases slight miosis was noticeable after 21 days, and in another two after 28 days. The rapidity of onset and the duration of miosis were directly proportional to the concentrations employed.

Maximal mydriasis was brought about in 10 normal eyes of five subjects by local instillation of one-percent atropine sulfate solution and in another 10 normal eyes of five subjects by four-percent homatropine hydrobromide solution. Single instillations of 0.1 cc. of 0.5-percent BC-48 abolished the action of atropine and homatropine in all these previously dilated eyes. Miosis was noticeable after one to three hours after instillations of BC-48 and reached its maximum after 24 hours. Miosis was maintained for five to seven days. The effects were more pronounced in homatropinized than atropinized eyes.

Maximal miosis was induced with 0.5-percent BC-48 solution in 20 normal eyes of 10 subjects. Single instillations of one-percent atropine sulfate six hours after BC-48 treatment abolished miosis in 10 such eyes and four-percent homatropine hydrobromide in the other 10 eyes. The effects were noticeable within 10 minutes after instillation of both atropine sulfate and homatropine hydrobromide.

Table 6 shows the effect of BC-48 on intraocular pressure of normal subjects. BC-48 lowered the intraocular pressure by 3.0 mm. Hg or more, and in two eyes more than 11 mm. Hg three hours after instillation. Although the tension-lowering effect was noticeable after 30 minutes in most eyes, in some during the first 30 minutes to one hour instead of lowering there was a transient rise of intraocular pressure. The maximum effect was noticed after 24 hours, when tensions reached their lowest point in all eyes. The tensions were still below their original values nine days after a single BC-48 instillation.

Tables 7 and 8 show the effect of BC-48 on the facility of aqueous outflow of normal human eyes. In all eyes there was a significant increase in the coefficient of aqueous outflow facility 24 hours after instillation.

TABLE 6

EFFECT OF BC-48 (DEMECARIUM BROMIDE) ON TENSION OF NORMAL HUMAN EYES

Patient	Eye	0.1 cc. %	Before Instillation	(Tension in mm.Hg [Schiøtz]) After Instillation						
				30 min.	1 hr.	3 hr.	5 hr.	24 hr.	5 da.	9 da.
E.R.	O.D.	0.1	17.19	14.72	14.72	14.72	12.55	12.55	12.55	14.72
	O.S.	0.25	19.99	19.99	14.72	14.72	14.72	12.55	12.55	14.72
A.K.	O.D.	0.1	13.60	14.72	12.55	12.55	10.65	10.65	10.65	12.55
	O.S.	0.5	13.60	12.55	12.55	12.55	10.65	10.65	10.65	10.65
F.B.	O.D.	0.25	12.55	14.72	14.72	12.55	8.99	6.30	8.99	12.55
	O.S.	0.1	14.72	10.65	10.65	10.65	8.99	6.30	8.99	8.99
M.P.	O.D.	0.25	28.72	26.75	23.17	19.99	17.19	10.65	10.65	12.55
	O.S.	0.5	23.17	23.17	23.17	19.99	19.99	12.55	14.72	14.72
R.P.	O.D.	0.5	13.60	12.55	10.65	10.65	10.65	10.65	10.65	10.65
	O.S.	0.1	13.60	14.72	10.65	10.65	10.65	10.65	10.65	12.55
W.G.	O.D.	0.5	26.75	14.72	12.55	12.55	12.55	12.55	14.72	14.72
	O.S.	0.25	23.17	17.19	12.55	12.55	12.55	12.55	14.72	14.72

Determinations of coefficient of aqueous outflow facility one and two hours after instillation showed some increase in facility in most eyes with the exception of two in which in one eye there was no change two hours after 0.5-percent BC-48 instillation and in the other an actual decrease one hour after 0.5-percent BC-48 instillation.

In all the normal eyes instillation of BC-48 was accompanied by symptoms and signs of ciliary spasm namely browache, blurred vision, increase in false myopia and increase in capacity for accommodation. In addition both superficial and deep congestion was noted in nearly all eyes, which lasted one to three days and was more marked when higher concentrations were employed. Two patients complained of nausea.

DISCUSSION

The cholinesterase inhibitors, when instilled locally into the eyes inactivate the cholinesterase, so that there occurs an accumulation of acetylcholine in the eye, which exerts its muscarinic action and probably accounts for the lacrimation, miosis, ciliary spasm, vasodilation and increased permeability of blood aqueous barrier, fall in intraocular pressure and increase in aqueous outflow facility.

Kraupp, Stumpf, Herzfeld and Pillat and Kraupp, Schwarzacher and Stumpf described a series of synthetic cholinesterase inhibitors belonging to the group polymethylene-bis-(N-methylcarbaminoyl-m-trimethylammoniumphenols) characterized by two neostigmine molecules connected by a polymethylene chain. By virtue of their chemical structure they possess great intensity and duration of action. Out of this group, BC-48 has been shown to be characterized by an intense and long duration of action on cholinesterase inhibition. BC-48 inhibits mainly the true cholinesterase or specific cholinesterase (contained in red blood cells) unlike DFP which chiefly inhibits the pseudocholinesterase or nonspecific cholinesterase (contained in plasma) as shown by Leopold and Comroe, or physostigmine and neostigmine which cause an approximately equal inhibition of true cholinesterase and pseudocholinesterase as quoted by Stumpf.

Herzfeld and Stumpf have shown that BC-48 has more prolonged action than physostigmine or neostigmine because of the great stability of the enzyme inhibitor com-

plex and because BC-48 is not split up by pseudocholinesterase. Leopold, Krishna and Lehman during the study of the effects of anticholinesterase agents on the blood cholinesterase levels of normal and glaucoma subjects have further shown that while both BC-48 and 217-MI inhibit the true cholinesterase mainly; in the case of BC-48 there is considerable inhibition of true cholinesterase after a short-term therapy or even following a single instillation, while in the case of 217-MI more time is needed to bring about this inhibition.

BC-48 is water soluble and is stable for indefinite periods in aqueous solution under ordinary conditions. Being a quaternary ammonium salt it penetrates poorly through the corneal epithelium, but by the same token it possesses certain inherent muscarinic properties. Thus according to Pillat, Stumpf, Gittler and Pommer, it exerts its action not only because of its anticholinesterase effect but also because of this inherent muscarinic action. Gittler and Pillat have confirmed this by showing that the low concentrations of BC-48 in the aqueous humor are sufficient to produce intense local muscarinic effects.

TABLE 7

EFFECT OF 0.1 CC. (TWO DROPS) 0.5 PERCENT BC-48 (DEMECARIUM BROMIDE) ON THE FACILITY OF AQUEOUS OUTFLOW OF NORMAL HUMAN EYES

Patient	Time after Instillation (hr.)	Coefficient of Aqueous Outflow Facility (in mm./min./mm. Hg)	
		(Control) O. D.	(Test) O. S.
C.C.	1	0.25	0.22
		0.22	0.19
E.G.	1	0.17	0.26
		0.17	0.29
A.G.	1	0.30	0.32
		0.25	0.33
V.V.	2	0.20	0.29
		0.21	0.29
F.C.	2	0.14	0.21
		0.16	0.25
R.L.	24	0.22	0.18
		0.22	0.29
D.T.	24	0.27	0.20
		0.24	0.32
N.M.	24	0.14	0.11
		0.14	0.30

TABLE 8

EFFECT OF 0.1 CC. (TWO DROPS) 0.25-PERCENT BC-48 (DEMECARIUM BROMIDE) ON THE FACILITY OF AQUEOUS OUTFLOW OF NORMAL HUMAN EYES

Patient	Time after Instillation (hr.)	Coefficient of Aqueous Outflow Facility (in mm./min./mm. Hg)	
		(Control) (O. D.	(Test) O. S.
S.H.		0.20	0.22
	2	0.18	0.27
	4	0.19	0.28
	24	0.22	0.31
A.P.		0.25	0.17
	2	0.23	0.29
	4	0.24	0.29
	24	0.23	0.30
C.W.		0.16	0.17
	24	0.19	0.28
W.E.		0.16	0.14
	24	0.15	0.32
C.H.		14.57	0.23
	24	14.57	0.29
J.P.		0.30	0.14
	24	0.28	0.18
L.O.		0.18	0.13
	24	0.18	0.18
M.R.		0.19	0.19
	24	0.19	0.23
P.C.		0.21	0.21
	72	0.24	0.33

Being a powerful cholinesterase inhibitor, BC-48 possesses powerful muscarinic and some nicotinic properties. Its actions are similar to those of neostigmine to which it bears some chemical resemblance. The present study on the side-effects of BC-48 in rabbits confirms this. In man the only systemic effect noted was nausea in two patients, after single instillation of 0.5 percent BC-48 solution into the eyes. Because of its increased permeability across blood-brain barrier and its powerful anticholinesterase effect, BC-48 could be very toxic.

The present study shows that BC-48 produces intense miosis, congestion of the eye both superficial and deep, fall in intraocular pressure and increase in the facility of aqueous outflow. It further shows that BC-48 produces blurred vision, false myopia, browache, periorbital pain and pain in the eye, and abolishes the action of atropine and homatropine and vice versa in normal human eyes. In some rabbits and normal human eyes instead of a decrease in intraocular pressure, there occurs an actual increase in initial stages. All these findings are in essential agreement with those of Gittler and Pillat. These effects are similar to those of DFP on rabbits' eyes as shown by Scholz and Sallmann and Dillon; DFP on normal human eyes as shown by Scholz and Wallen and Leopold and Comroe; 217-MI on rabbits' eyes as observed by Krishna and Leopold; and 217-MI on normal human eyes as shown by Leopold, Gold and Gold. These effects are more pronounced in their intensity and duration with BC-48.

Finally the deduction arrived at by Gittler and Pillat that physostigmine when given first, blocks the action of subsequent instillation of BC-48 from their clinical observations of failure of control of glaucoma by BC-48 in patients who were treated with physostigmine before, is not borne out by our experimental and clinical observations. We have studied the synergisms and antagonisms between BC-48 and other anticholinesterase agents in rabbits after the method employed by Koster in studying synergisms and antagonisms between physostigmine and DFP. Our studies show that physostigmine when given first blocks the action of subsequent doses of DFP as observed by Leopold and McDonald and of 217-MI as observed by Krishna and Leopold but no such blocking mechanism was demonstrated between physostigmine and BC-48.

Summary

1. BC-48 is a very potent cholinesterase inhibitor. It inhibits mainly the true cholinesterase.

2. BC-48 possesses powerful muscarinic and some nicotinic properties. It resembles neostigmine in action.

3. BC-48 is readily soluble in water. It is stable indefinitely under ordinary conditions.

4. BC-48 does not cause any permanent damage to normal rabbit and human eyes when instilled locally. It produces intense miosis, ciliary spasm, vasodilation and increased permeability of the blood-aqueous barrier, lowering of intraocular pressure and increase in the facility of aqueous outflow.

5. BC-48 is not blocked by physostigmine. It seems to be synergistic with other anticholinesterase agents.

1601 Spring Garden Street (30).

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CORNEAL LENSES IN OPHTHALMIC PRACTICE*

HOWARD AGATSTON, M.D., ALAN H. BARNERT, M.D., AND MORRIS FELDSTEIN, M.D.
New York

INTRODUCTION

A doctor today cannot ignore contact lenses. The manufacturers, as a result of long research, have developed lenses that can be worn eight or more hours daily by 70 to 80 percent of all wearers. Over 50 percent wear them all day long. Improved fitting techniques are also responsible for the sharp increase in the number and percentage of satisfied patients. One company alone recently filled orders for over 100,000 lenses in one month. This renewed public interest and acceptance of the lenses is reaching enormous proportions. It is creating a major problem in eye care.

ROLE OF THE OPHTHALMOLOGIST

How are we responding to this new problem and what role do we play? This varies with the individual ophthalmologist. It may also vary from patient to patient. The types of professional service we now render may be classified as follows: Consultation, prefitting examination, examination during and after fitting by a technician, and fitting by the ophthalmologist.

1. *A consultant.* An opinion is given often without benefit of routine examination. If the patient is strongly motivated to have lenses for cosmetic and psychologic reasons alone, he may be encouraged by some doctors and discouraged by others. There is a difference of opinion here. The patient may ask to be referred to a contact lens company or to a technician for fitting. Some ophthalmologists are reluctant to recommend lenses, let alone learn to prescribe them, because they fear that the cornea will be damaged or that a comfortable and successful fit is unlikely.

2. *Prefitting examination.* In addition to the role of consultant, the ophthalmologist is often asked to perform a routine examination before fitting is begun. If we find that the eyes are healthy and there are no contraindications to contact lenses we give our approval. Some contact lens companies require this type of limited medical clearance.

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3. *Examination during and after fitting by a technician.* This may be done on the advice of the ophthalmologist or at the request of the technician or the patient. It may or may not have been preceded by the consultation and prefitting examination. The doctor checks the refraction with the contact lenses in place. After the lenses are removed and fluorescein instilled he checks the corneas under the slitlamp. The lenses must be worn five to 10 hours before this examination is done.

4. *Examination and lens fitting by the ophthalmologist.* He refracts, takes the necessary measurements, sends the prescription directly to the manufacturer and obtains the lenses. When the lenses arrive he checks them for accuracy. He examines the patient with the lenses in place and he checks the cornea after the lenses are removed. The sequence of examination may be summarized as follows: Examination of patient, examination of lens, examination of lens or cornea, and examination of cornea and lids after lens is removed. The doctor and his office assistant teach the patient to insert, reposition, and remove the lenses.

Fitting requires attention to more clinical details than a routine refraction. It necessitates external examination and biomicroscopy, as well as accurate interpretation of symptoms. Because of his training and scientific background the ophthalmologist is eminently qualified to participate in this work. Many of our colleagues have acquired or are acquiring a practical knowledge of contact lenses. More and more take an active part in fitting their patients. They realize that this work requires clinical skill and that it is not primarily a laboratory procedure.

Basic theory of corneal lenses

By a *corneal lens* we mean any lens which is made to fit over the cornea alone and not over the sclera; this includes lenses of varying sizes, shapes and details of construction, such as the microlens, the contour lens, and many others, as well as the somewhat larger Tuohy-type corneal lens. In contrast, a *scleral lens* has a corneal part, for optical purposes, and a part which rests on the sclera.

In fitting a contact lens the criteria for proper fit are (1) absence of damage to the cornea, and (2) the comfort of the patient; comfort including absence of symptoms and proper correction of the refractive error.

To attain these criteria there are two main factors involved—the fit and the refraction; we will discuss these. Some of the other factors, which will not be discussed, are such things as outside bevel, edge treatment, character of surface, properties of the material, thickness, grooves, holes, depressions, and so forth.

The *fit* is mainly concerned with the looseness or tightness of the lens, and this in turn depends on (1) the back surface or base curve, (2) the overall size of the lens, called diameter, and (3) the amount and character of the bevel, if any. Each of these will be discussed.

The *refraction* of the lens is determined by the power of the contact lens itself and also by its relation to the cornea, since this forms a so-called lacrimal lens which affects the refraction of the lens-eye combination.

Factors affecting fit

1. Base curve

The back curve or base curve of the lens, can be either flatter than the cornea, steeper than the cornea, or concentric with the cornea, which is called "on K"; (K for keratometer reading). The flatter the lens the looser it is; a flat lens slides loosely on the cornea, while a steep lens grips it. If a lens is too tight or too loose, therefore, one way of correcting this is to replace it with a new lens having a flatter or steeper base curve. It is not practicable to grind a new base curve on an existing lens. In general the other factors are varied rather than the base curve to change the looseness or tightness.

Note also, that the tears behind the con-

tact lens form a lens of their own, which is convex in the case of a steep lens and concave in the case of a flat lens, so the base curve has an effect on power as well as on fit. This will be discussed later.

The back surface is actually a portion of a sphere, and is measured by the radius of that sphere of which it is a portion; this is called radius; so the radius of a lens is the radius of the base curve; base curves are about seven to eight mm. The base curve may also be measured by the equivalent number of diopters; for instance a 7.50-mm. base curve can be called a 45 diopter one.

The base curve of a lens can be determined by putting it on a keratometer, and taking a reading. Keratometers are usually calibrated in both mm. of radius and in diopters.

The flatness or steepness of the base curve in relation to the cornea on which it rests can also be determined by putting the lens on the eye and then putting in a drop of fluorescein. The eye is then examined with a strong blue light so that the layer of fluorescein between the lens and the cornea fluoresces yellow-green. Depending on the relation between the lens and the cornea, various patterns will be seen; where the layer is thick it will appear light; where it is thin it will be dark. The light and dark areas show whether the lens touches in the periphery (steep) or at the apex (flat) and similar facts.

2. Size

The second factor in fit is the size of the lens, which is called diameter. It is measured in millimeters on a micrometer or more easily on a special diameter gauge. The effect on tightness is this: the bigger the lens is, the more it grips the cornea and so the tighter it is. A small lens, other factors being equal of course, is looser.

The reason for this depends on an anatomic fact: the central portion of the cornea is considerably steeper than the periphery; the cornea is paraboloid. The back curve of a lens is therefore steeper relative to the periphery than it is relative to the apex, and increasing its size therefore has the effect of making it steeper and therefore tighter. A given lens can be made looser by grinding some off the edge; naturally a lens cannot be made larger except by replacing it with a new one.

3. Bevel

The third factor influencing the looseness and tightness is bevel, also called peripheral curve. A lens may have a small amount of the back surface cut away near the edge; this is the bevel. The sharp junction between the two curves is usually or always smoothed off or blended. In some lenses with a large bevel a second bevel is ground in between the peripheral curve and the main curve, or optical portion. This curve is called the intermediate or secondary curve. The bevel is sometimes intended to clear the cornea and sometimes intended to match the peripheral portion of the cornea, which is flatter than the central portion. But in any case the effect of the bevel is to make the lens looser than it would be without it. The bigger the bevel, the looser the lens. A lens can be made looser by cutting more bevel out.

Clinically, the looseness or tightness of a lens is judged mainly by (1) lag and (2) symptoms.

By *lag* is meant the slight sliding motion of the lens on the cornea. To determine lag, the patient is asked to look in various directions. (Or the examiner may move or displace the lens by pushing the lower lid against it. The examiner may also ask the patient to blink.) Under these conditions the lens will move away from its position on the cornea; this is called lag, and the amount of the motion and speed of return to its normal position are indications of fit. A lens which is tight will move little or not at all; a lens which is loose will slide around more. The proper amount is difficult to describe and must be learned by experience.

To summarize the fit then, a flatter, a

smaller or a more beveled lens is looser; a steeper, or a larger, or a less beveled lens is tighter. Various combinations of these factors can be, and are used to fit the same eye; which combination gives the best overall results in each type of case is gradually being learned.

About the refraction, there is not much to say. It involves (1) the power of the lens itself and (2) the lacrimal lens.

The lens itself is simply a plus or minus sphere, no cylinder being ground on it. The power can be measured on a lensometer.

The lacrimal lens is the layer of tear fluid between the contact lens and the cornea; depending on the relation of the anterior corneal surface to the back surface of the lens it may act as a minus, a plano or a plus lens. It is not an important factor in lenses fitted on "K," but in flat lenses it may be −2.0 or −3.0 diopters, or more.

Since the lacrimal lens bathes the anterior surface of the cornea in fluid having almost the same refractive index as the cornea, it almost eliminates the cornea as a refracting surface and thus automatically neutralizes any corneal astigmatism whether regular or irregular, which may be present. Corneal astigmatism therefore may be disregarded from an optical standpoint when corneal lenses are worn. Lenticular astigmatism on the other hand cannot ordinarily be corrected by corneal lenses; fortunately this is rarely a problem.

In ametropia of over five diopters the vertex distance becomes a factor which must be considered when ordering the lens.

History

The history of corneal contact lenses can be divided into two periods, with 1948 as the dividing line. While there is considerable reference to corneal lenses prior to 1948, going back as far as 1888, these lenses were not actually in practical use. The clinical use of corneal lenses received tremendous impetus with the advent of Tuohy lens in 1948. The chief characteristic of this lens was that it was fitted flatter than the corneal curvature. The lens was about 11.4 mm. to 12 mm. in diameter, and had a peripheral bevel of about 1.5 mm. Patients using this lens had an average wearing time of from four to six hours and only about 30 percent of the patients fitted with it were reasonably satisfied, in our experience.

In 1954, the so-called microlens was produced. Like the Tuohy lens, this was also fitted flatter than the corneal curve, but was considerably smaller, being 9.5 mm. in diameter and not bevelled at the periphery. Only about 50 percent of the patients trying this lens could be successfully fitted. They had an average wearing time of about eight hours.

In 1955, Norman Bier of England devised the contour lens. The basic concept of this lens was that it was fitted to conform with the corneal curve as closely as possible throughout the entire area of the lens. As originally devised, this lens was 9.65 mm. in diameter, and had an optical zone 7.0 mm. wide. A secondary curve which was flatter than the central optical curve was ground into the periphery of the lens. With this lens, the average wearing time was from eight hours to all day, and it was reported that about 80 percent of all patients trying this lens were successfully fitted.

Since then, there have been numerous other lenses of various types, some of which will be simply listed for information. The Hornstein lens is a lens in which the central area is fitted steeper than the corneal curve, and the peripheral area conforms to the corneal curve. The Stimson lens has four projecting facets on the inside surface of the lens on which the lens rests. The Vent-Air lens has four grooves ground on the inside periphery, spaced at 90 degrees from each other. Bifocal corneal lenses have also been devised. There are two types. One type has a peripheral curve ground on the outside surface, and the other type has a central depression ground on the inside surface. Another lens utilizes the idea of a two mm. clear opening in the center of the lens with

the rest of the lens opaque. This is to provide a greater depth of field by diaphragming down the optical system.

Some attention has been paid to methods of measuring the corneal surfaces for the purpose of prescribing more accurately the peripheral curvatures of lenses. A recent method which has been described is called photoelectric keratometry. This consists of a photograph of reflection of a series of concentric rings from the cornea. The picture is then scanned with a special photoelectric and computing machine which gives the data to describe the corneal radii of curvature throughout its entire area. While this method has been used by its inventor, it is not available for practical use.

A special method of finishing the edges of the lens is called "conlish" edging. This simply standardizes the method of finishing the lens and provides for a very smooth edge.

In the fitting of difficult cases, one group in Chicago is trying the effect of hypnotism. The results seem to be promising according to their reports, but this is still purely experimental.

In the manufacturing of lenses, there are some experiments going on with the idea of creating a large number of perforations in a lens by using a stream of high speed electrons. In Europe there is a group experimenting with the use of the corneal lens for constant wear, even while the patients are asleep. There are other investigators who are using the corneal lenses in cases of glaucoma, presumably with benefit to this condition. There are unconfirmed reports about the use of corneal lenses to slow down or prevent the progression of myopia. Corneal lenses are also being used in cases of esophoria and esotropia. And finally, corneal lenses are now widely used for refractive correction in monocular and binocular aphakia.

Indications

Contact lenses have a number of advantages over conventional spectacles.

1. *First there are cosmetic and psychologic advantages.*

Young ladies gain in self confidence and poise when they can discard their spectacles and still retain normal visual acuity. Matrons and women in the business world also appreciate their new appearance. The psychologic effect is similar to that obtained following successful strabismus surgery on an adult.

2. *Increased field of vision* is notable in high refractive errors and in aphakia, and is important in a number of activities, such as ballet dancing, sports and motoring. The increased visual field afforded is of particular advantage in twilight and night driving.

3. *Better visual acuity* is obtained in cases of irregular astigmatism, keratoconus and refractive errors in the presence of nystagmus.

4. *Less aniseikonia* results in correction of monocular aphakia—eight to 11 percent instead of 30 percent. In anisometropia of two or more diopters the greatest discomfort is not from aniseikonia, but due to the artificial heterophoria created whenever the eyes move from the primary position. With a two-diopter difference in optical correction one prism diopter of deviation is incurred if the gaze deviates five mm. from the optic axis. Sometimes these difficulties may be overcome by the use of contact lenses. This is another advantage. They do not create heterophoria.

5. *Magnification in myopia* of moderate and high degree is an optical advantage.

6. *No fogging* of lens by mist, rain, snow, mud or dust.

7. *Safety factor* compared to eyeglasses.

Summary of Indications

1. *Refractive errors* of 1.5 or more diopters, particularly when glasses are in constant use. The cosmetic and psychologic values are greatly appreciated by some. Others are more impressed by the optical advantages. In nystagmus with refractive error, contact lenses are properly centered in all positions of gaze. When the eyes are turned to the position of minimal nystagmus, there-

fore, contact lenses will give better visual acuity than conventional glasses.

2. *Aphakia.* In monocular aphakia aniseikonia is reduced. In monocular and binocular aphakia there is increased field and no prismatic distortion. A +3.0D. contact lens in the nonaphakic eye neutralized with spectacles may eliminate the residual aniseikonia.

3. *Keratoconus* is an indication for corneal lenses. Occasionally the scleral lenses must be resorted to.

4. *Irregular astigmatism* whether it follows inflammations, trauma, corneoscleral surgical wounds or corneal grafting.

5. *Therapeutic.* A contact lens with an opaque periphery may be employed as an antiglare measure in aniridia and albinism. Contact lenses may be used cosmetically to cover disfiguring corneal scars.

6. *Vocational and avocational indications* may be briefly summarized by listing such activities as acting, singing, dancing, flying, motoring, sports, mining, deep-sea diving, and seafaring. Corneal lenses are becoming popular with high school and college athletes particularly in basketball, baseball, football, track and tennis.

From the list of avocations and vocations one might conclude that contact lenses are primarily used by people in active physical pursuits. Actually those who appreciate lenses most are the ones whose work is at near range—reading, desk work, and so forth.

Contraindications to the use of corneal lenses

Contraindications to the use of corneal lenses are quite numerous:

1. The most important of them is the absence of a clear indication for their usage. We have already described the indications for the use of corneal lenses. If these are absent, the patient should be advised not to have the lenses fitted.

2. A second group of contraindications revolves around personality factors. There are numerous patients whose personal motivation is insufficient. An example is the child who is urged by a doting parent to have corneal lenses fitted but who himself does not really like the lenses. Such a case is better rejected before starting.

3. Those cases in which epiphora is at all marked. These cases cannot be fitted well, since the lenses tend to float around on the excess tear fluid.

4. Excessive dryness of the eye due to a variety of clinical conditions. Some of these conditions may be vitamin-A deficiency, Sjøgren's syndrome, or ocular pemphigus. In general, any condition which produces excessive dryness of the eye should be reason for rejection.

5. Ocular infection of the lid, conjunctiva or cornea.

6. Ocular allergies, again of the lid, conjunctiva or cornea. Of course, if these infections and allergies can be treated and removed, the cases may then be considered suitable for corneal lens fitting.

7. Local neoplasm. Examples of these would be dermoids, pigmented nevi, or granulomas.

8. Degenerations of the cornea, such as corneal dystrophies and band keratitis. One might possibly include those cases with recurrent erosions of the cornea under this classification. However, whether this is called a dystrophy or degeneration, the history of recurrent erosions of the cornea is sufficient reason for rejecting the case.

9. Corneal edema. For the time being, our knowledge of the reaction of an edematous cornea under a corneal lens is insufficient to warrant use of these lenses in such instances.

10. A high degree of lenticular astigmatism. Since the corneal lens is unable to correct this condition, the patient would be left with a fairly poor vision. The corneal lenses are therefore unsatisfactory for such cases.

11. Marked exophthalmos. Such patients are very uncomfortable and contact lens fitting is usually unsuccessful.

12. Special lid conditions. There is the type of patient who has thick, tight lids. A little experience with the fitting of these lenses will soon show one how to recognize

such cases so that they may be rejected at the onset. Other special lid conditions which are causes for rejection are entropion and trichiasis.

13. Corneal insensitivity, as seen in neuroparalytic keratitis and trigeminal nerve resection. Since these patients cannot receive any sensory warnings of corneal irritation, they should not be fitted with corneal lenses.

14. Presbyopia. While this condition is not in itself a real contraindication to the fitting of corneal lenses, the patient may feel a bit discouraged to find that he is unable to read without an extra pair of glasses to be worn over his corneal lenses. However, there are numerous patients who are aware of this limitation at the onset and prove to be very satisfactory patients for corneal lenses. In this connection, one must again bring up the question of bifocal corneal lenses. Although they do exist, they are not easy to fit and are not altogether satisfactory. At the present time, they are not advisable for routine usage. They are still in the experimental stage.

In this connection it might be mentioned that hyperopes near the presbyopic age do well with contact lenses, but older myopes who are comfortable reading with spectacles may be less comfortable with contact lenses and may need a reading add. Near-presbyopic myopia therefore is a relative contraindication. (The reason for this is optical and has to do with effectivity.)

Office procedure

This description of the fitting procedure obviously cannot go into all the details, since there are variations in details from patient to patient. The routine to be outlined will present an average routine rather than a system which one must rigidly follow.

The first visit takes one half to three quarters of an hour. It is devoted mainly to a search for indications and contraindications for contact lenses, evaluation of the patient and his motivation, as well as measurement for the lenses. The first visit, therefore, consists of taking a history, during which an evaluation of the patient is made, a complete eye examination including refraction, and a special examination which includes (1) a careful keratometry, which is the most important figure needed for ordering, and (2) an estimation of the palpebral fissures, corneal diameters and average pupil size; these may modify the type of lens ordered.

After this is done, stock lenses may be inserted, if desired, in order to show the patient how they look and feel, how they are inserted and removed, and so on; and possibly to check the figures of the keratometry and refraction. We do not consider trial lenses an important or basic part of fitting; we believe that they are, at most, a minor adjunct and should not be used instead of or in conflict with the keratometer reading.

When the examination is finished, the doctor discusses with the patient the pros and cons of contact lenses in his case, what he may expect from them, and so on. The lenses may be advised, advised against, or strongly discouraged. Accordingly the patient may order the lenses or give up the idea. Or, in many cases, he may go home to think about it. This ends the first session.

If the patient orders the lenses, either at that time or later by telephone, it may have to be decided what lens type to use, and what the diameter, the bevel, the radius (which is the back curve) and the power will be. For those unfamiliar with ordering, it is simpler to send the keratometer reading and the refraction to the laboratory where a standard type lens can be calculated and sent to you.

When the lenses arrive you should check them. The diameter is checked with a special diameter gauge. The power is checked on an ordinary lensometer, making sure at the same time that no accidental prism is in the lens. The thickness can be checked with a special thickness gauge. The width of the bevel is checked with a magnifying glass. The radius of the back surface is carefully checked on the keratometer.

The patient then returns for the second session which takes about an hour. About half of this hour can be spent with the nurse

or assistant. After topical anesthesia, the lenses are put on. A general idea of the fit is obtained by looking at the lag and by checking the fluorescein pattern. Checking vision with plus and minus spheres over the lenses gives a general idea of the refraction. We say general idea of fit and power because these things change as the lenses are worn. There is usually more tearing at the beginning and both the fit and the power may not be the same at first as they are later so, unless a gross error is found, no new lenses are ordered at this time.

After the checking, the patient gets his first lesson in insertion and removal of the lenses. The nurse may do this. The first lesson takes some time. In addition to teaching the patient, the positions and maneuvers which are easiest and most effective for him have to be worked out. (For instance some patients prefer to use the right hand for each eye while others prefer the right hand for the right eye and the left hand for the left eye.)

At the third session, which takes about 15 minutes of the doctor's time, the lenses are inserted and the patient wears them for about an hour in the waiting room. The patient then is checked for fit and power, as before. In addition, slitlamp examination is made after the instillation of fluorescein. Then the patient practices, with or without local anesthesia, insertion and removal, checking and reviewing what he has learned. He is also taught how to replace the lens onto the cornea in case it should slip off into the fornix, or in case it is inserted off the cornea, as may happen.

The fourth session is similar to the third, except that no anesthesia is used.

After the third or fourth session, if the patient is proficient in inserting, removing, and repositioning his lenses, he can take them home and start wearing them. Wearing time is increased by one hour a day until the lenses can be worn for four or five hours; then wearing time is increased by two hours a day until they can be worn all day.

The patient is seen once a week for several weeks, then every two weeks. At these visits, he is asked about symptoms, especially halo or blur, tearing, falling out, and wearing time. The lag and fluorescein pattern are examined. The cornea is examined for stain, epithelial edema and, sometimes, for change in keratometer reading. The refraction is checked as before. If at any time the lens has to be changed for fit or power, it is. This, however, is rare.

If the original figures were correct and the lens was made according to them, it is not often necessary to change it; many lenses are changed unnecessarily. Some conditions requiring a change in the lens and their diagnoses are:

1. *Lens too loose.* This is determined by excess lag, central staining of the cornea, a history of the lens falling out. It is corrected by ordering a larger lens, or one with less bevel, or one with a steeper base curve.

2. *Lens too tight.* Determined by too little lag, symptoms of halo or burning (from production of epithelial edema) or change in refraction or keratometer reading after wearing the lenses. It is corrected by decreasing the diameter, increasing the bevel, or ordering a flatter lens.

3. *Lens too flat.* If a fluorescein pattern shows that corneal abrasion has been produced, a steeper lens should be ordered.

4. *Lens too steep.* This is determined by a fluorescein pattern. A too-steep lens may produce the same symptoms as a too-tight lens. It is corrected by ordering a flatter lens, or, in some cases, by increasing the bevel.

5. *Wrong refraction.* Diagnosis and correction are obvious.

A certain amount of tearing, hyperemia, foreign-body sensation, photophobia, and other symptoms are normal during the first few hours of wearing the lenses. If they persist after that, the polish and edges of the lenses should be inspected and corrected, if necessary. If this does not help, it may be that the patient cannot wear contact lenses.

The fitting should be complete by about

three months; the patient should be followed every four months thereafter.

Pitfalls

We have included pitfalls because it is important to acknowledge and learn from one's mistakes and to learn from the mistakes of others.

Case selection is not always easy and one cannot accurately foretell success. Most strongly motivated patients should be given a chance to wear lenses. Those with weak motivation should be discouraged or told to go home and think it over. A few notable successes can make a doctor overoptimistic and overenthusiastic. There may be a tendency to oversimplify the procedure and in this way to interest people whose need is doubtful or whose motivation is weak. All the contraindications to the wearing of contact lenses must be studied and the patient must be told that a period of adjustment is unavoidable.

Naturally most of the pitfalls occur during the fitting procedure. The refraction is usually accurate but vertex distance, lacrimal lens and the transposition of the prescription to minus cylinders must be taken into consideration. The ophthalmometer or keratometer must be standardized regularly, at first daily and later weekly. This is done by use of a standard steel ball. The axis of the astigmatism should be recorded so that the keratometer reading corresponds to the flattest meridian and steepest meridian. The technique of measuring corneal diameter should be such as to minimize inaccuracy due to parallax. Exact readings should be obtained when the lensometer is used. The lens should be clean and dry. The end-point must be sharp.

One of the most common pitfalls is inability to differentiate adaptive symptoms from those of a poor fit. A tight lens may cause sufficient tearing to make the lens appear loose. Adaptive symptoms are considered normal and they are the result of a foreign-body reaction to the lens. This is to be expected. It consists of tearing, lid irritation, blinking, inability to look up, and blurring of vision. Adaptive symptoms usually subside in a few days if lenses are worn two or more hours a day. Another common pitfall is failure to check on the technique the patient uses in the care of the lenses and in their insertion, repositioning, and removal.

Improper criteria for fitting a lens satisfactorily may be a serious pitfall. Many patients who wear contact lenses full time and who are quite happy and comfortable show chronic superficial corneal staining with fluorescein. Apparently the corneas are partially anesthetized as a result of repeated trauma. Since the ophthalmologist cannot overlook chronic corneal staining, a more suitable fit must be found or the patient forbidden to wear corneal lenses. After the fitting procedure is completed, the eyes must be checked three or four times a year to rule out corneal trauma.

Finally, one should avoid fitting or prolonging the fitting of an unsuccessful patient. It is a dreadful waste of time and it may create bad feelings. Although the contraindications may be observed, it may be impossible to avoid this pitfall. The patient's motivation may be only temporary or high astigmatism may interfere with a satisfactory outcome.

Comment

Now that corneal lenses constitute an important part of eye care, they should be fitted by someone who knows both lenses and eyes. Up to now the majority of contact lenses have been fitted by nonmedical persons. Medical supervision has either been absent or limited to a preliminary examination. This is undesirable from many points of view. A technician's approach to fitting tends to be a mechanical one. Medical implications are overlooked. And especially, fitters have no experience with corneal lesions.

In contrast to the tremendous amount of work on contact lenses themselves, they have

undergone very little real clinical investigation. There are a large number of similar lenses with various trade names but the only evaluation of respective merits and disadvantages has been the number of orders received.

Every ophthalmologist should know how to fit corneal lenses so that he can either fit them himself or actively supervise others. Every ophthalmologist should know all about the use of contact lenses so that he can advise his patients properly. Courses to train ophthalmologists in contact lenses fitting should be established. Residents should be trained in this field just as they are trained in refraction and motility. Contact lenses clinics should be established for training and research as much as for furnishing lenses.

In short, contact lens fitting is and should be an integral part of ophthalmic practice.

35 East 35th Street (16).
960 Park Avenue (28).
1225 Park Avenue (28).

COMPLETE OR PERIPHERAL IRIDECTOMY IN CATARACT SURGERY

C. DWIGHT TOWNES, M.D.
Louisville, Kentucky

Because my associates and I had a general impression that eyes upon which complete iridectomy was done during removal of cataracts progressed through quieter convalescent periods to results practically as satisfactory as those achieved in eyes upon which peripheral iridectomy was performed (by peripheral iridectomy we mean the excision of a good-sized piece of iris near its root, leaving a coloboma which extends at least halfway to the pupil; performance of peripheral iridotomy was abandoned several years ago), we decided three years ago to accumulate equal series of both types of iridectomy, each series being large enough to provide a valid comparison.

In choosing subjects for this study, we excluded cases of congenital, traumatic and complicated cataract, those of senile cataract with coexistent primary glaucoma, and those in which the lens capsule ruptured and was not completely removed. We established no special criteria by which choice of peripheral or complete iridectomy was determined, except that in eyes with rigid pupils which were not dilated beyond five mm. on preoperative examination, complete iridectomy was done. If a patient's second eye was being operated upon, the type of iridectomy performed upon the first eye was usually repeated; however, if complications had been encountered in the first eye, complete iridectomy was done at the second operation.

There were 45 patients who had bilateral peripheral iridectomy, 39 who had bilateral complete iridectomy and 19 who had one peripheral and one complete iridectomy. A few of these eyes are not included in this study because they were operated upon several years earlier with different techniques, making them unsuitable for comparison. Distribution as to the age, sex, type and maturity of lens changes are quite similar in both groups, as shown in Tables 1, 2 and 3 which, we believe, present representative and unbiased findings.

TECHNIQUE OF OPERATION

All of the operations were done by me. Every patient underwent physical examination before operation, either by his family physician or by the resident staff in the hospital upon admission the day before surgery.

TABLE 1

TYPE OF LENS OPACITY

	Immature			Mature		Hypermature	Total
	Nucl	Cort.	Brun.		Brun.		
Group A. Complete							
Noncomplicated	17	42	2	39	1	10	111
Complicated	10	25	1	27	3	3	69
Group B. Peripheral							
Noncomplicated	15	32	0	34	3	3	87
Complicated	15	29	2	33	5	9	93

Preliminary sedation consisted of Nembutal (1.5 gr.) at bedtime the night before operation. Sodium amytal (3.0 gr.), Demerol (50 to 100 mg. intramuscularly, depending upon age, sex and weight) and Thorazine (25 mg. intramuscularly) were administered one and one-fourth hours before operation. When any contraindication to barbiturates existed, Phenergan (25 mg.) was substituted.

Intravenous sedation was administered to about 60 of these patients, when they were unduly apprehensive or gave promise of being recalcitrant. This consisted of intravenous injection of a mixture containing Thorazine (25 mg.), Phenergan (50 mg.) and Demerol (100 mg.) in normal saline (25 cc.). It was given slowly until the patient fell asleep. The average dose required was approximately seven cc. of the mixture.

Anesthesia and akinesia were obtained by instillation of Ophthaine (0.5 percent) or Pontocaine (0.5 percent), by a modified Van Lint injection of the lids and by a retrobulbar injection using Novocaine (4.0 percent) or Xylocaine (2.0 percent) containing adrenalin (1:50,000) and Wyadase (6.0 units per cc.)

An incision in clear cornea was used in all operations. Grooves approximately 0.5-mm. deep were made at the 11- and 1-o'clock positions just inside the superficial marginal plexus, and a tiny conjunctival flap was dissected free at the site of each groove. Sutures (6-0 chromic gut) were placed, one needle at this time also going through the conjunctival flap. A small keratome incision was made at the 12-o'clock position and enlarged with scissors almost to the horizontal meridian.

The iridectomy was performed. No mydriatic was used before surgery because it was much easier to do either peripheral or complete iridectomy with the pupil undilated. (Occasionally, when a mydriatic is instilled beforehand, the pupil will be maximally dilated so that it is not possible to grasp the iris with forceps.) For peripheral iridectomy the iris was grasped near its root, withdrawn partially and a good-sized portion excised. For complete iridectomy, the iris was grasped halfway between its root and the pupil.

TABLE 2

AGE

	31–40	41–50	51–60	61–70	71–80	81–90	91–	Total
Group A. Complete								
Noncomplicated	1	11	19	40	29	11		111
Complicated	1	4	7	22	27	7	1	69
Group B. Peripheral								
Noncomplicated		9	18	28	29	3		87
Complicated		4	17	33	32	7		93

TABLE 3

SEX DISTRIBUTION

	Male	Female	Total
Group A. Complete			
Noncomplicated	58	53	111
Complicated	28	41	69
Group B. Peripheral			
Noncomplicated	35	52	87
Complicated	41	52	93

withdrawn until the pupil margin could barely be seen, the scissors brought from below, the iris carried between the blades of the scissors toward the center of the cornea and the iris cut. This will insure a high percentage of narrow, straight colobomas. One drop of adrenalin (1:1,000) from a sterile ampule was dropped on the iris and the pupil promptly dilated.

Delivery of the lens was accomplished by phakoerisis with a motor-driven pump providing continuous suction.

When the sutures were tied, the second needle was brought through the conjunctival flap so that a bit of conjunctiva was included in the knot. It had been found that 6-0 chromic gut was absorbed very slowly when used to close corneal incisions. Inclusion of conjunctiva in the knot hastens absorption. Usually the sutures are gone by the time the patient makes his first visit to the office about 15 days after surgery. The ends of the sutures were anchored to conjunctiva about eight mm. above the limbus. This was insurance against the knot coming untied and prevented the long ends from sweeping over and irritating the cornea. If pigment clouded the anterior chamber it was removed by irrigation with normal saline.

Air was injected only rarely when there was no indication of immediate reformation of the anterior chamber. The wound was inspected carefully and, if the edges of the incision were not properly coapted a supplemental suture was inserted. This was necessary in only a few eyes. Atropine was instilled, a monocular dressing applied and the patient kept in bed only one day.

COMPLICATIONS

Of the 180 eyes having complete iridectomy (Group A) 111 developed no complications referable to removal of the cataracts during the period of observation, which varied from five to 40 months; 69 eyes developed one or more complication, totaling 92 complications. In the peripheral iridectomy group, (Group B) 87 eyes have so far showed no complications, while 93 eyes developed a total of 141 complications.

Table 4 lists the complications encountered. Heading the list are the cases in which there was rupture of the hyaloid, adherence of the hyaloid to the corneal wound, and anterior synechias, with or without eversion of the iris. There were 39 such eyes in Group A and 55 in Group B.

The next listing shows the eyes which had rupture of the hyaloid accompanied by macular degenerative changes, resulting in varying degrees of reduction of visual acuity. While some of these eyes did not present typical ophthalmoscopic evidence of macular degeneration, no other cause of the lowered visual acuity could be determined. This is the type of complication so well described and documented by Irvine.[1]

TABLE 4

COMPLICATIONS ENCOUNTERED

	Group A Complete	Group B Peripheral
No complications	111	87
Ruptured hyaloid Adherent hyaloid Anterior synechias	34 (5)*	35 (20)
Ruptured hyaloid with macular degeneration	(8)	(11)
Vitreous loss	2 (1)	1
Uveitis	8 (2)	19 (3)
Detached retina	4	7
Glaucoma	1	2 (6)
Hyaloid contact	1	(1)
Pigment clump	8	7 (1)
Detached choroid	6 (2)	12
Delayed reformation	3 (1)	3 (3)
Hyphema	2 (2)	7 (3)
Gaping wound sutured	(2)	
	69 (23)	93 (48)

* Figures in parentheses indicate eyes which also are listed under other categories.

In my opinion, the kind of iridectomy performed would seem to have no relation to loss of formed vitreous. Vitreous loss may, however, be due to faulty technique on the part of the surgeon, except when the vitreous is completely fluid as it was in one of the eyes in Group A. Loss of such vitreous is unavoidable.

Uveitis, in the severe form, can be a most distressing complication. Beginning with deep-seated pain about the fourth to sixth day, presenting chemosis of the conjunctiva, murky aqueous with many cells, posterior synechias, sometimes an exudative membrane across the pupil and numerous vitreous opacities, it is slow to respond to therapy. Use of steroids, topically and systematically, is definitely effective in shortening the course of the disease.

Five of the 10 eyes in Group A, and 10 of 22 in Group B developed severe uveitis. Almost half of these eyes failed to recover visual acuity up to 20/40. The only eye in this entire series requiring enucleation was one with peripheral iridectomy which developed intractible uveitis and glaucoma.

Detachment of the retina was observed four times in Group A. Three of these cases occurred early, 30, 46 and 62 days after operation. None was cured by retinal detachment surgery. The fourth detachment occurred seven months after cataract removal and was cured by scleral resection.

In the eyes of Group B retinal detachment occurred seven times. One detachment was seen in the immediate postoperative period and was believed to have existed prior to cataract surgery. Detachment surgery was not performed in this case. This patient's other eye was the one reported in the preceding paragraph as being cured.

The other six detachments occurred at intervals of 110 to 1,000 days after cataract removal. Three have been cured. One of these patients, after having both cataracts removed one week apart, suffered retinal detachment in the right eye 110 days later. This was operated upon without success. Retinal detachment in the left eye occurred 295 days later. Simple retinopexy replaced the retina with visual acuity of 20/20. Twenty months later retinal detachment recurred in another area. Scleral resection resulted in replacement for five months when detachment recurred, involving three fourths of the retina. Diathermy applied around a break in the areas of the scar from previous surgery replaced the retina; it has remained in place to the present.

It is extremely interesting to me that glaucoma has been observed in only one eye in Group A. The pressure became elevated to 60 mm. Hg (Schiøtz) when the hyaloid was formed and was not adherent to the wound. Upon treatment with pilocarpine and Diamox, the pressure gradually was reduced to normal. All treatment was stopped after four months. When last seen, two years after the cataract operation, pressure measured 14 mm. Hg (Schiøtz). The hyaloid had reformed and was not adherent to the wound.

In Group B, eight eyes developed glaucoma. Two of these may be cases of primary open-angle glaucoma, although secondary glaucoma cannot finally be ruled out. The other six cases are secondary to peripheral anterior synechias from shallow anterior chambers with detached choroid, or from hyaloid adhesions, either ruptured or intact.

Vitreous contact with the cornea occurred once in each group. In Group A, after rupture of the hyaloid on the 31st day in an eye which had cornea guttata, strands of vitreous were adherent to the cornea. Air injection into the anterior chamber broke up the adhesions temporarily but later a few thin strands again became adherent.

The eye in Group B had a normal hospital convalescence but, when seen in the office on the 15th postoperative day, there was a flat chamber, the hyaloid was in contact with the cornea which was moderately cloudy, and there was a large detachment of the choroid. A tight pressure dressing was applied and the patient sent to the hospital to have a posterior sclerotomy and air injection. However,

when she came to the operating room several hours later the anterior chamber had reformed and the cornea was clearing. No surgery was done and the eye improved under pressure dressing.

Pigment clumps on the cornea occurred in equal numbers in the two groups. Fortunately in only one eye was the clump near enough to the center of the cornea to interfere with vision.

In addition to clumps on the cornea, slitlamp examination revealed moderate or numerous pigment granules adherent to iris, hyaloid, and cornea in a rather high percentage of eyes in both groups: 28.1 percent in Group A and 37.2 percent in Group B. It is probable that delivery of the lens through the intact pupil dislodges more pigment, as does the movement of the pupil in the postoperative stage. While these pigment granules disappear in time, they may block the trabeculae and therefore can not be considered innocuous.[1] The anterior chamber should be irrigated following removal of the cataract whenever there is doubt as to its being clear.

In my experience, detachment of the choroid rarely occurs during the hospital convalescent period but usually after the patient goes home, as the result of some sort of trauma even though trivial, such as sitting down too hard on a chair or bumping into furniture or a door frame. Ordinarily a tight pressure dressing and bedrest will restore the anterior chamber and reduce the detached choroid. Careful attention should be given to the anterior chamber. It should not be allowed to remain completely empty longer than four or five days. It should be determined whether the loss of the chamber was due to a leaking wound or to an iris block, and measures should be instituted to re-establish it. In Group A it was necessary to resort to operative treatment two times.

The same caution should be used in delayed reformation of the anterior chamber, which was not of frequent occurrence or serious import in either group. If the incision has been closed meticulously, the anterior chamber will begin to reform before the patient leaves the operating table. Accurate closure of the incision, with supplemental sutures where the wound edges are not in good apposition, is essential in producing a watertight wound quickly. This complication occurred four times in Group A and six times in Group B. Surgical intervention, consisting of air injection into the anterior chamber, was necessary in only one eye.

The incidence of hyphema is reduced greatly when the incision is made wholly in the cornea. It occurred four times in Group A, and 10 times in Group B. No case was serious and all hyphemas were absorbed quickly.

Two eyes in Group A presented slight gaping of the wound between the two sutures at the 11- and 1-o'clock positions. In neither case was the anterior chamber lost. A supplemental suture was placed in each case. Although one of these eyes later developed detachment of the retina, it had recovered well in the meantime.

State of hyaloid

Noncomplicated eyes

Table 5 indicates the form of the anterior hyaloid observed with the slitlamp postoperatively in noncomplicated eyes. Seventy-one (63.9 percent) of the eyes in Group A presented hyaloids which were smooth and flat or smooth with a slight bulge, while 40 (36 percent) showed the hyaloid to be loose and wavy, sometimes surging back and forth through the pupil, or to be herniated, like a mushroom of varying size, into the anterior chamber. In Group B the same forms of the hyaloid appeared 46 times (52.8 percent) and 41 times (47.2 percent) respectively.

Complicated eyes

Table 6 shows that the hyaloid ruptured in 30 eyes of Group A and in 42 eyes of Group B. The rupture was observed at the time of

TABLE 5

STATE OF HYALOID IN NONCOMPLICATED EYES

	Flat Smooth	Smooth Sl. Bulge	Loose Wavy Surging	Small Mushroom	Large Mushroom	
Group A Complete	28 (25.2%)	43 (38.7%)	18 (16.2%)	10 (9%)	12 (10.8%)	111
Group B Peripheral	17 (19.5%)	29 (33.3%)	13 (14.9%)	14 (16.15%)	14 (16.15%)	87

the first postoperative visit, nine times in Group A and 13 times in Group B. Later ruptures occurred after the hyaloid was noted to be flat or slightly bulging, four times in Group A and nine times in Group B. Rupture followed surging or mushrooming hyaloid, 17 times in Group A and 20 times in Group B.

These observations suggest that the hyaloid is less likely to rupture when it remains smooth and flat or slightly bulging after cataract removal and rupture is more likely to occur when the hyaloid is loose and surging or when it mushrooms into the anterior chamber. It is my belief that when the hyaloid is smooth and flat or slightly bulging, the posterior hyaloid not detached, the whole vitreous body being in a fairly healthy state, the anterior hyaloid is less likely to rupture. It seems that when the hyaloid is loose and surging or herniates far into the anterior chamber, the posterior hyaloid is often found to be detached and retracted forward and the vitreous is partially liquefied. In this kind of eye, the hyaloid ruptures most often.

Hilding[2] has pointed out that movements of the vitreous may be profoundly altered following intracapsular cataract extraction. Also, the adherent strands which form with or without rupture of the hyaloid further modify movement of the vitreous and can be important factors in the etiology of retinal detachment and macular changes.

VISUAL RESULTS

From Table 7 we see that, in Group A, 107 eyes, including 32 which suffered complications, obtained visual acuity of 20/25 or better. In addition, 48 eyes, 20 with complications, had visual acuity of 20/30 or 20/40, making a total of 155 eyes which recovered comfortable reading vision.

In Group B, 114 eyes, including 46 which had complications, recovered visual acuity of 20/25 or better, and 37 others (25 with complications) could see 20/30 or 20/40.

Thus, 85 percent of the 360 eyes recovered visual acuity of 20/40 or better. This figure is not as high as I should like to see it.

The fact that 123 of these eyes suffered and recovered from complications, sometimes multiple, is incontrovertible evidence that the

TABLE 6

STATE OF HYALOID IN COMPLICATED EYES

	Intact	Ruptured	Reformed	Adherent	Anterior Synechias
Group A Complete	39	30*	10	23	16
Group B Peripheral	51	42†	10	27	23

	*	†
On first postoperative office examination	9	13
Following smooth flat or slightly bulging hyaloid	4	9
Following mushrooming or surging hyaloid	17	20

TABLE 7

VISUAL RESULTS

	20/20 20/25	20/30 20/40	20/50 20/70	20/80 20/200	Less than 20/200	Blind	
Group A. Complete							
Noncomplicated	75 (41.6%)	28 (15.5%)	5	2	1	0	111
Complicated	32 (17.8%)	20 (11.1%)	7	3	4	3	69
Total	107 (59.4%)	48 (26.6%)	12	5	5	3	180
Group B. Peripheral							
Noncomplicated	68 (37.8%)	12 (6.6%)	2	4	1	0	87
Complicated	46 (25.5%)	25 (13.9%)	12	3	2	5	93
Total	114 (63.3%)	37 (20.5%)	14	7	3	5	180

eye is a pretty tough organ, able to withstand and overcome the effects of injury and diseases to a very remarkable degree.

ANALYSIS OF OBSERVATIONS (table 8)

Comparison of these two groups was carried out by applying the test of the significance of differences in proportions.

The most important information to be gained from Table 7 is that regarding the difference in incidence of complications in the two groups. In this comparison, P = 0.007, meaning that only seven times in one thousand would such a difference occur from chance alone. Since this is a rare occurrence it can be reasonably concluded that the difference is not a chance difference, but a real one due to the difference in the type of iridectomy performed.

When the specific complications are compared, only glaucoma and uveitis fall into a range of probability which is significant. However, the actual figures in both instances are so small that the comparison is less meaningful.

Relative to ruptured hyaloid, adherent hyaloid, and anterior synechias, in which comparison P = 0.053, significance is borderline, being a little above the accepted figure.

In no other comparisons was the probability within a significant range.

SUMMARY

A series of 360 operations for removal of senile cataract has been presented. It consisted of two groups alike in all respects except for the type of iridectomy performed. In Group A, complete iridectomy was done; in Group B, peripheral iridectomy.

The two groups were compared as to complications and visual results.

Significance tests were applied to the observations obtained.

TABLE 8

ANALYSIS OF OBSERVATIONS BY SIGNIFICANCE OF DIFFERENCE IN PROPORTIONS

	Complete Group A	Peripheral Group B	Relative Deviate	Probability
No complications	111	87	2.7	P=0.007
Glaucoma	1	8	2.31	P=0.02
Uveitis	10	22	2.23	P=0.026
Ruptured hyaloid, Adherent hyaloid, Anterior synechias	39	55	1.93	P=0.053
Hyphema	4	10	1.66	P=0.097
Delayed reformation	3	6	1.00	P=0.32
Detached choroid	8	12	0.91	P=0.36
Detached retina	4	7	0.91	P=0.36
V.A. 20/20–20/25	107	114	0.76	P=0.45
V.A. 20/30–20/40	155	141	0.58	P=0.56

Conclusions

1. This study appears to bear out the impression that eyes operated upon with complete iridectomy in cataract extraction suffer fewer complications and progress through quieter convalescent periods than do eyes upon which peripheral iridectomy is done.

2. There is no statistically significant difference in visual results obtained by the two types of iridectomy.

1015 Heyburn Building.

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EFFECT OF EARLY AMBULATION*

On the incidence of postoperative complications of cataract surgery

Norval E. Christy, M.D.
Taxila, Pakistan

Introduction

In the past 10 to 15 years there has been a great change in the postoperative care of patients who have undergone cataract operations. Formerly extreme care was exercised to prevent any movement, or at least any straining, on the part of the patient. Four to six attendants were required to move the patient from the stretcher to his bed; one or two attendants were required to assist the patient in any movement in his bed; and the patient was kept still in bed for a week or 10 days.

The development and use of more secure methods of corneoscleral wound closure and the demonstration by general surgeons of the benefits of early ambulation in preventing such complications as pneumonia, phlebitis, and loss of muscle tone and strength, particularly in older patients, have emboldened ophthalmologists to allow their patients to get out of bed sooner and to engage in more postoperative activity.

With the large number of methods of wound closure and the variety of materials recommended for corneoscleral wound closure, it is impossible for the average ophthalmic surgeon to try more than a few techniques or suture materials in any significantly large series of cases. It is also difficult for him to know what part of his success with any given method is due to the excellence of that particular method and what part is due to the natural ability of the eye to heal without complication when it is handled gently and when the wound edges are approximated and allowed to heal. It is also difficult for him to know without risk to a certain number of patients what the effect of varying amounts of postoperative activity might be on his results, using a particular method of wound closure.

This series of 3,000 cases is presented to show the effect of varying amounts of postoperative activity on the incidence of immediate postoperative complications in patients having only the simplest of wound closure. It is felt that, although the findings may not be directly applicable to a series of cases using some other method of wound closure, still one might expect the changes in incidence of complications to follow a similar pattern.

Materials and Background

The 3,000 cases reported here were all treated in a simple village hospital catering

* From the American Mission Hospital.

to the poor in northern Pakistan during a two-year period. In Pakistan, because of weather conditions and social custom, eye surgery, at least in village areas, is seasonal. During the summer season when the temperature rises to 110-115°F. patients prefer not to lie in bed in a hospital if they can avoid or postpone it, since almost none of the hospitals are air-conditioned. In the winter when the temperature may drop toward freezing, patients requiring elective surgery again avoid the hospital since some hospitals, including ours, are not adequately heated.

Another factor affecting the seasonal variation is that most of the patients are farmers whose main crop is wheat. As soon as the wheat crop is ready for harvest in the spring there is a great exodus from the hospital so that every able-bodied man, and some of the women too, may help with the harvest. Even when the patient is too old to be of much help in the fields the person who has accompanied him to the hospital leaves and takes the patient with him.

All this results in a great fluctuation in patient census. In slack seasons there may be less than 20 patients in the eye department of our hospital and in busy seasons the number may be over 400.

This fluctuation raises the problem of how to maintain a nursing staff adequate to care for 400 patients and still keep this staff occupied when the patient load drops to 20. There is no pool of surplus nurses in Pakistan from which nurses might be hired temporarily in the rush seasons. The problem is partly solved in our hospital, and in some others, by requiring each patient to bring with him to the hospital a relative or friend whose job it is to help with simple nursing care—feeding, toilet needs, bathing (if any), and so forth. The patient's more specific medical needs are taken care of by a small staff of trained nurses. This system of using lay nursing help has resulted in a considerable degree of variation in the quality of care given to patients and in the amount of freedom allowed the patient in the matter of postoperative ambulation.

For many years it was our rule to require patients to lie in bed for eight days after a cataract operation. The strictness with which this rule was enforced depended somewhat on each patient's lay helper. Some were very solicitous of the patient's welfare and enforced the rule exactly; others through carelessness or because of the imperiousness of some tyrannical old patriarch (or matriarch) patient almost completely disregarded the rule.

Then, too, almost every season one or two beggars would demonstrate their independent unregimented natures by getting out of bed and going off to the bazaar to beg on the day of operation and on every subsequent day. It was noted with some surprise that such independent activity seldom, if ever, resulted in disaster or even damage to the operated eye. This experience plus a report of experience with early ambulation of patients having had cataract surgery by the late Mr. W. Seymour Philps of London[1] inspired the study here reported.

Methods

Three groups, totaling 3,000 operated cases, were studied. All operations were performed by the same surgeon under the same conditions and using practically the same technique. (Changes in technique are noted below.) The patients were almost all Pakistani villagers. The distribution of cataract types among the three groups was very similar and was roughly as follows: immature, seven percent; mature senile, 61 percent; intumescent, 12 percent; hypermature, 11 percent; morgagnian, 0.5 percent; juvenile, 3.5 percent; traumatic, one percent; and complicated, four percent.

The first group of 1,000 cases were operated upon consecutively in the spring of 1957 and were required to stay in bed for eight days following operation. After the operation these patients were lifted from the operating table to a wheeled stretcher by

means of a cloth stretcher placed beneath them on the table before the operation. They were wheeled about 75 yards to their beds and lifted into their beds by two or three stretcher bearers.

The second group of 1,000 consecutive cases performed in the fall of 1957 were transported from the operating table to their beds in the same way but were required to stay in bed only 24 hours.

The third group of 1,000 cases performed in the spring and fall of 1958 were assisted in climbing down from the operating table and then walked the 75 yards to their beds. These patients were allowed to be out of bed as they wished. In all cases only the operated eye was bandaged. In 10.6 percent of the cases both eyes were operated upon on the same day. These patients also walked to their beds.

The surgical technique was roughly the same as that described in a previous article.[2] Patients were seen for the first time in an out-patient clinic in the afternoon and were then admitted for surgery the following morning. Patients were given 1.5 gr. of phenobarbital the night before operation and 3.0 gr. of phenobarbital and 25 mg. of chlorpromazine on the morning of operation. After the intraocular pressure had been measured, O'Brien and Van Lint blocks were given, using two-percent procaine, and a retrobulbar injection of two cc. of two-percent procaine with six units of hyaluronidase was given. A cotton traction suture was placed in the upper lid and a speculum introduced.

The incision was made at the limbus with a von Graefe knife to include the upper one half of the cornea. At the superior part of the incision a bridge of conjunctiva about two-mm. wide and six-mm. long was left intact. An iridectomy was performed and the lens removed beneath the conjunctival bridge. After removal of the lens, the iris was smoothed back into place as necessary, the wound was inspected to see that edges were smoothly in apposition, and the conjunctival bridge smoothed in place. Penicillin ointment and sulfa powder were placed in the conjunctival sac, the eye was closed, the lid suture taped down on the cheek, and a dry pad applied. As many as ninety operations were performed in one morning.

Two significant changes in technique were instituted during these series. The type of iridectomy was varied as follows: in the first 1,000 cases peripheral iridectomies were performed in 36.5 percent; in the second 1,000 cases peripheral iridectomies were performed in 69.6 percent; and in the third group of 1,000 cases peripheral iridectomies were done in 50.3 percent. The percentage of intracapsular extractions varied as follows: in the first group 67.2 percent, in the second group 76.1 percent, and in the third group 77.8 percent. Linear extractions for juvenile or traumatic cataracts were performed in 4.5 percent of all cases.

Results

The following complications were recorded:

1. Vitreous was lost at operation in 2.5 percent, 3.1 percent and 2.0 percent of the cases in the three groups.

2. Blood was seen in the anterior chamber on the first dressing (first postoperative day) in 11.9 percent of the cases having eight days bedrest, in 10.3 percent of the cases having one day's required bedrest, and in 11.9 percent of the cases who walked from operating table and had no required bedrest.

3. Late hyphemas developed in 5.0 percent of the first group, 5.5 percent of the second group, and 4.9 percent of the third group.

4. Transient striate keratitis was seen in 19.4 percent of the first group, 20.5 percent of the second group, and in 24.8 percent of the third group.

5. There was some delay in the formation of the anterior chamber in 6.3 percent of the cases in the first thousand cases, in 12.3 per-

TABLE 1

EFFECT OF EARLY AMBULATION ON POSTOPERATIVE COMPLICATIONS OF CATARACT SURGERY

Group	Degree of Activity	No. of Cases	Complications (in percent)														
			Vitreous loss	Early hyphema	Late hyphema	Striate keratitis	Delayed formation of anterior chamber	Nonformation of anterior chamber	Cortex	Capsule	Iris prolapse	Iris incarceration	Infection	Choroidal hemorrhage	Conjunctival edema	Bulging wound	Secondary suturing	Secondary needling	Good vision	Useful vision
1	8 days bedrest	1000	2.5	11.9	5.0	19.4	6.3	1.7	7.8	7.5	1.2	1.4	0.1	0.5	2.1	0.4	0.2	1.2	91.6	2.9
2	24 hours bedrest	1000	3.1	10.3	5.5	20.5	12.3	2.3	4.8	5.4	2.0	1.9	0.7	0.5	2.6	0.3	0.0	0.1	91.0	1.9
3	No bedrest required	1000	2.0	11.9	4.9	24.8	9.3	3.5	4.4	5.1	1.8	2.9	0.1	0.4	1.4	1.0	0.2	1.0	91.6	2.4

cent of the second thousand, and in 9.3 percent of the third thousand.

6. The anterior chamber was still flat on the eighth postoperative day in 1.7 percent of the cases in the first group, in 2.3 percent of the cases in the second group and in 3.5 percent of the cases in the third group.

7. Cortex was noted in the anterior chamber in 7.8 percent of the cases in the first group, 4.8 percent of the second group, and 4.4 percent of the third group.

8. Capsule was visible to the naked eye in the anterior chambers of 7.5 percent of the cases in the first group, 5.4 percent of the second, and 5.1 percent of the third. In connection with these two complications it should be noted that of the first thousand operations 29 percent were capsulotomies, of the second thousand 18.5 percent were capsulotomies, and of the third thousand operations 19.4 percent were capsulotomies.

9. Iris prolapse was noted in 1.2 percent of the cases in the first group, 2.0 percent of the second, and 1.8 percent of the third group.

10. Iris incarceration without prolapse but sufficient to distort the pupil was noted in 1.4 percent of the cases in the first group, in 1.9 percent of the cases in the second group, and in 2.9 percent of the cases in the third group.

11. Infections occurred in 0.1 percent of the cases in the first group, in 0.7 percent of the cases in the second group, and in 0.1 percent of the cases in the third group. Six of the seven infections in the second group could be traced to a disastrous error in the preparation of a solution used in the eyes.

12. Choroidal hemorrhages occurred in 0.5 percent of the cases in groups one and two and in 0.4 percent of the cases in group three.

13. Conjunctival edema occurred in 2.1 percent of the cases in the first group, 2.6 percent of the cases in the second group, and 1.4 percent of the cases in the third group.

14. Bulging wounds were noted in 0.4 percent of group one, in 0.3 percent of group two, and in 1.0 percent of group three.

15. It was necessary to do a secondary suturing of two wounds in the first group (0.2 percent) and of two wounds in the third group.

16. A secondary needling was done in 1.2 percent of the cases in group one, in 0.1 percent of the cases in group two, and in 1.0 percent of the cases in group three.

These data are shown graphically in Table 1.

There was no significant difference in the visual results in the three groups. By the rough methods used in testing early postoperative visual acuity among these illiterate patients 94.5 percent of the patients in group one, 92.9 percent of those in group two, and 94 percent of those in group three obtained good or useful vision.

Discussion

In this study of 3,000 cases radically increasing the amount of ambulation and activity following cataract surgery did not result in a radical increase in the complications even though corneoscleral wounds were not sutured. The wound edges were held in apposition only by a bridge of conjunctiva about two-mm. wide and by a firm eye bandage. Operations were done with a minimal amount of instrumentation and manipulation of the eye. Small increases were noted in the incidence of striate keratitis, delayed formation and nonformation of the anterior chamber, bulging wounds, and iris incarceration. It is noteworthy that there was no significant increase in the incidence of iris prolapse, early or late hyphema, nor choroidal hemorrhages and that the visual results were almost identical in the three groups of cases.

Summary

Data concerning the incidence of complications following cataract extraction in 3,000 cases are presented. The cases were divided into three groups of 1,000 each with varying amounts of postoperative activity permitted in the three groups. One group was required to stay in bed for eight days; a second group was required to stay in bed for one day; and the third group walked from the operating table to their beds and were permitted to move about as they wished.

In all cases the corneoscleral wounds were held closed only by a bridge of conjunctiva and an eyepad. The operations were performed quickly and with a minimum of instrumentation and trauma of the eye.

The radical increase in the amount of postoperative activity did not result in any radical increase in postoperative complications. No significant increase was noted in the incidence of early or late hyphema, iris prolapse, or choroidal hemorrhages. The visual results in the three groups were almost identical.

American Mission Hospital.

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EMOTIONAL GLAUCOMA*

Maurice Croll, M.D., and Leo J. Croll, M.D.
Detroit, Michigan

The role of emotion as a causative factor in acute closed-angle glaucoma is not clearly understood nor has it been fully evaluated. Unfortunately the type and titer of emotion cannot be measured by a tonometer; the only evaluation comes from clinical experience.

In 1929, Inman reported acute glaucoma following emotional distress of which the patient was totally unaware. Duke-Elder

* From the Department of Ophthalmology, The Grace Hospital.

states "attacks of glaucoma . . . are infrequently induced by conditions which upset the vasomotor balance and are particularly associated with worry, sleeplessness, fatigue or constipation."

Schoenberg, in 1940, stressed the role of anxiety states in the pathogenesis of primary glaucoma. He stated, "mental and emotional conflicts, whether conscious or buried in the unconsciousness may produce a disturbance of physiologic processes and even cause irreversible organic lesions."

Egan, in 1955, reported four cases which he called "shock glaucoma" and related each one to a fall. He elaborated further, ". . . shock glaucoma should include those attacks ushered in by emotional upsets as well as those caused by distant physical trauma, or both, or indeed whether such a separate designation is unwarranted at all, remains for the future to reveal."

It is quite clear that emotional upsets from a great variety of causes play a role in closed-angle glaucoma. The mechanism of exactly how this occurs remains to be explained and needs more accurate observations, more exact records, detailed histories of each minute attack, and directed inquiry of the anxiety states and emotional status of the patients. One may have to probe for the last two factors if their role is to be properly evaluated.

Ripley, a psychiatrist interested in glaucoma, kept a record of intraocular pressures on a patient during various emotional stimuli. His records on this one patient covers a period of 31 months (fig. 1) a fairly representative time. It is an excellent study of the relationship of increased ocular tension in direct relationship to various stimuli. Many more detailed studies of this type are needed before definite conclusions can be made.

Is there a type of personality which when subjected to acute emotional episodes will have closed-angle glaucoma in preference to an ulcer, headache, migraine, or gastrointestinal upset? Two basic components are necessary for production of an emotional type of glaucoma—a pre-existing shallow an-

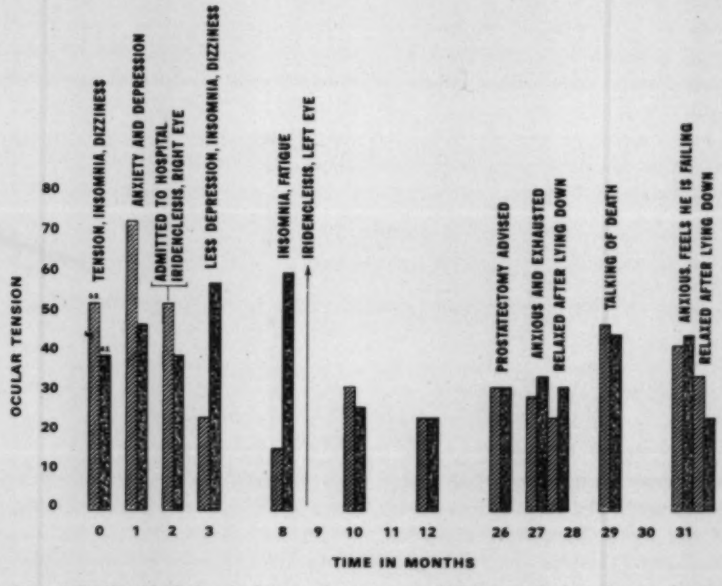

Fig. 1 (Croll and Croll). Variation in intraocular pressure (McLean) during changes in environmental situations and emotional reactions in a patient who was under observation for 31 months. (After Ripley.)

terior chamber and a superimposed acute emotional episode.

A shallow anterior chamber is found in about 10 percent of the population. Studies seem to indicate that this is an inherited characteristic. A shallow anterior chamber angle may be closed off for a great variety of reasons which can be divided into five main groups: (1) anatomic, (2) functional, (3) cerebral, (4) drugs, and (5) trauma to the eye.

Anatomic factors

There are four main anatomic factors that contribute to the progressive narrowing of the anterior chamber: (1) gradual narrowing of the anterior chamber with advancing age, (2) progressive continuous growth of the lens through life, (3) pupillary block by a dilated pupil, and (4) swelling of the vitreous.

With advancing age the anterior chamber becomes progressively and increasingly more shallow. Rosengren in an excellent treatise demonstrated this by actual measurement. An eye already hypermetropic, shorter, and smaller than normal provides an excellent ground structure for closed-angle glaucoma. Rosenberg emphasized that the shallow anterior chamber is a preexisting fact and is not the result of increased intraocular pressure. He stated, ". . . the difference between the depth of the chamber demonstrable between normal and glaucomatous eyes exists before the pressure increases and has been shown to be independent of the increase."

Tørnquist in a monograph entitled, "Shallow anterior chamber in acute glaucoma: A clinical and genetic study," measured the depth of anterior chambers. He found a decrease from 3.19 mm. (in the 20-year age group) to 2.68 mm. (in the 65-year age group), a relatively large percentage decrease with advancing age.

Kronfeld measured the actual quantity of aqueous in glaucomatous eyes as compared with normal eyes. He found the total volume in glaucomatous eyes to be from 50 to 100 cu. mm. as compared to 200 to 250 cu. mm. in normal eyes.

The lens continues to enlarge all through life. The diameter increases from 8.67 mm. in the 20 to 29 age group to 9.49 mm. in the 60 to 69 age group. In the same period it increases sagittally from 4.0 mm. to 4.77 mm. This increase represents almost one mm. in both diameters. As a result, the shallow anterior chamber becomes further embarrassed by an increase in the lens in both size and volume.

The only true mechanical block that exists in the eye is that of a dilated pupil. Whatever the cause may be, the iris is crowded more and more into a shallow chamber and again there is a good anatomic basis for the precipitation of an attack of acute closed-angle glaucoma.

In taking histories an attempt should be made to trace any transient blurring of vision, ocular discomfort, pain or presence of halos to see if they can be related to a dilated pupil. It would be rewarding to acquaint a patient with the nature of these episodes, put him on guard and have him observe the size of the pupils, as well as any symptoms that may occur at the same time. He should be advised that, if visual symptoms persist for more than 20 minutes, he should report to his doctor immediately.

Acute closed-angle glaucoma attacks are periodic, occurring predominantly in women, and so-called minor attacks tend to recur with increasing frequency, becoming progressively more severe in degree. That is why detailed, minute records are so vital.

What part does the vitreous play in this type of glaucoma? The vitreous, having the major volume, can hardly be a bystander while the rest of the eye is acutely and seriously threatened. Priestly Smith, in 1888, found that an increase of one mm. Hg pressure in the vitreous resulted in almost complete obliteration of the anterior chamber. Von Graefe attributed the shallow anterior chamber to high pressure in the vitreous chamber.

How is this brought about?

Evans was of the opinion that the aqueous flow posteriorly was facilitated by the cleavage planes present in the vitreous. These "cleavage planes" are channels which are remnants of the embryonic vascular system. Under acute emotional stress we know that the skin capillaries blanch and that similar changes take place in the cerebrovascular system. Is it possible for such changes to occur in the choroidal vessels?

Cholst has presented experimental evidence that such circulatory changes, violent in degree, take place in the eye under shock conditions. Repeated choroidal thickening with inadequate posterior drainage may also be a factor in precipitating the many minor episodes of short duration which are so characteristic of closed-angle glaucoma.

When vasodilation occurs in the posterior segment of choroid, there is an increase in volume of the vitreous as well as back pressure; as a result, the aqueous leaves the anterior chamber at a rate more rapid than normal. The iris is then carried anteriorly and pushed forward into the angle, thus closing it. Again, it should be emphasized that this chain of events could occur only in already abnormally narrowed angles. In adequate angles this type of attack could not be precipitated.

These four important anatomic factors can operate singly, in groups, or all together. They all have one common factor, however. They can, individually or collectively, embarrass an anterior chamber angle which is already shallower than normal.

Aqueous

Formation and circulation

After the aqueous is formed by the anterior uvea, the ciliary body, it flows into the posterior chamber, through the pupil, and into the anterior chamber. Its course continues through the canal of Schlemm to the aqueous veins.

The functions of aqueous are manifold. It maintains the normal range of intraocular pressure necessary for optical purposes. It furnishes nourishment to the lens and to some extent the cornea since these structures are devoid of blood vessels. It carries oxygen to the interior structures of the eye and carries away the products of metabolism, including carbon dioxide to the blood.

The normal blood-aqueous barriers are distributed to three areas of the eye. In the posterior portion the barrier consists of the capillaries of the retina; in the central portion (a) the walls of the uveal capillaries, (b) the anterior portion of the retina and its prolongations, (c) the ciliary epithelium, and (d) the epithelium of the posterior surface of the iris; in the anterior portion of the eye the blood-aqueous barrier is the naked uveal vessels on the anterior surface of the iris. The intraocular fluid is formed by diffusion from the endothelial walls of the capillary walls (fig. 2).

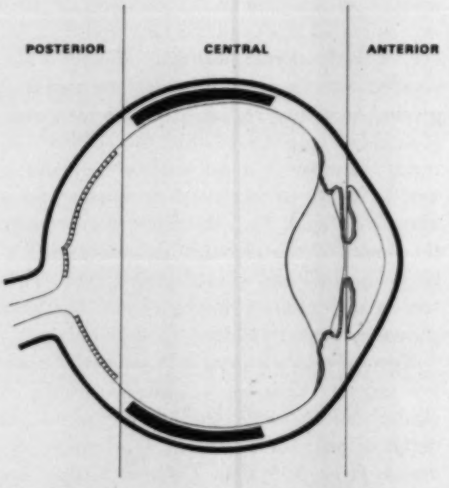

Fig. 2 (Croll and Croll). Barriers of the eye (diagrammatic). Posterior portion: retinal capillary walls. Central portion: (1) walls of uveal capillaries, (2) anterior portion of retina and its prolongations, (3) ciliary epithelium, (4) epithelium of posterior surface of iris. Anterior portion: anterior surface of iris (the naked uveal capillaries).

Dynamics of the aqueous

What factors in aqueous dynamics cause elevation of intraocular pressure in narrow-angle eyes?

When the volume of the globe is increased by a sudden influx of blood dilating the choroidal bed, the intraocular pressure is immediately increased, assuming, of course, that the sclera is rigid and unyielding and the outflow drainage channels are blocked by an embarrassed narrow angle. The intraocular pressure may also be increased when the venous outflow is blocked in part or in full. In the first instance the normal rate of outflow is diminished; in the second the entire outflow is dammed back. This set of circumstances is usually triggered by increased pressure in the episcleral vortex veins blocking the outflow of aqueous.

The trabecular outlet may become blocked for several reasons: edema of the trabecular meshwork, pigmentary deposits, collagenous degeneration and sclerosis of the trabeculae. Any of these pathologic conditions can cause diminished aqueous outflow, the degree depending on the extent of impairment.

According to Kinsey's concept of aqueous dynamics (fig. 3), in addition to the normal outflow by way of the canal of Schlemm, there is also a continual loss of the fluid through the structures which surround the anterior chamber. Water goes back into the vessels of the iris and into the cornea. Nonelectrolytes diffuse back into the vessels of the iris also. Electrolytes on the other hand do not diffuse back into the iris vessels.

Central control of intraocular pressure

Diurnal pressure variations and the interrelationship and dependence of the circulatory effects in one eye with those of the other strongly suggest the presence in the central nervous system of a mechanism for regulating intraocular pressure. However, there is much to learn about its exact location and function, its motivation, what influences it and its relationship to the general vascular system.

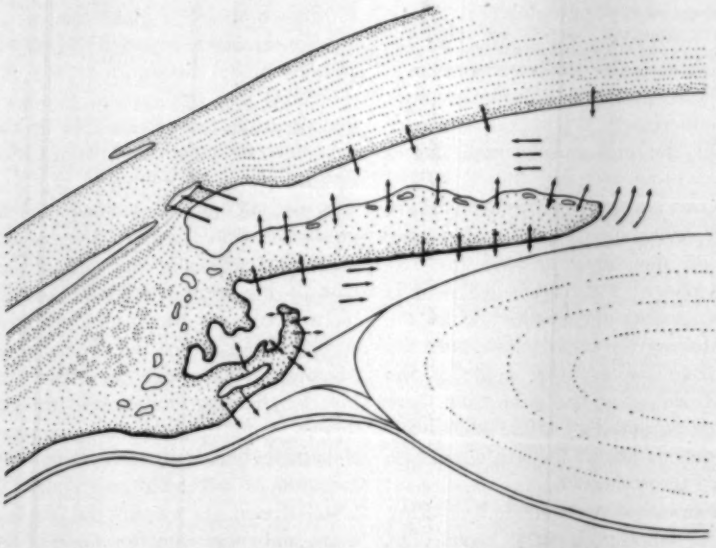

Fig. 3 (Croll and Croll). Kinsey's scheme, showing the dynamics of the formation of aqueous humor as found in the anterior chamber.

All of us have, however, had the experience of seeing a case of acute closed-angle glaucoma precipated by an upheaval in the patient's emotions. The death of a loved one, some type of physical injury, surgery or trauma, acute anxiety states may trigger an attack of acute closed-angle glaucoma. That the incidence of such attacks is not more frequent would seem to indicate that the trigger mechanism can only be set off in an eye with a shallow chamber. Such emotional crises seem incapable of affecting eyes with normal anterior chambers.

Role of the pupil

The role of the pupil in acute closed-angle glaucoma is closely tied in with any emotional stress on the central regulating mechanism. It may be that this relationship is the key to the etiology of this type of glaucoma. There are certain basic facts regarding the pupil that enter into the clinical picture.

The pupillary blocking syndrome consists of two key mechanisms: pupillary dilatation and pupillary block. There is a definite relationship between pupil size and production of increased tension. Acute glaucoma can be precipitated by dilating the pupil. For example, if a patient with shallow chambers is put in a dark room, the intraocular pressure may become increased. When the patient returns to light, the tension is lowered, due to miosis.

Actually, two antagonistic mechanisms are involved in acute narrow-angle glaucoma. Dilatation of the pupil crowds the iris into the periphery of the angle where it is held snugly against the meshwork of the trabecula. Making the pupil miotic pulls the iris away from the drainage angle of the trabecula. However, at the same time there is an increase in pupillary block due to collection of aqueous behind the iris, again lifting it toward the trabecula.

Actual observations were made by placing susceptible patients in a dark room. The pressure in these eyes was immediately elevated. Smith of the Eye Institute of London examined this type of eye with a gonioscope. The angle was apparently open, at once throwing doubt on the entire concept. However, when he turned on the light, the angle appeared closed but opened promptly with pupillary constriction. This is evidence for closed angle during dilation and open angle during constriction of the pupil.

What is the trigger mechanism that sets off the whole chain of events by dilating the pupil?

In times of stress, rage, excitement, and acute anxiety, the pupil dilates, how widely depends on the nature, degree, and duration of the stimulus. During stress suddenly imposed on the body the adrenal medulla liberates varying amounts of adrenalin. The pupil also dilates when the sympathetics are stimulated, with concomitant inhibition of the third nerve. Stimulation of the hypothalamus also causes a discharge of adrenalin into the blood stream. The three mechanisms acting together cause an accumulative effect and, as a result, the pupil can dilate quite widely. In susceptible eyes with narrow angles, only an additional stimulus is needed to precipitate a full-blown attack of glaucoma.

Jaffe, in 1949, reported the experimental psychosensory dilatation of the pupil. He concluded that this type of pupillary dilatation occurs in two phases. The first is purely on a nervous basis, due to stimulation of the sympathetics and inhibition of third nerve. The second phase, somewhat delayed, is purely hormonal and is determined by the amount of adrenalin liberated. He concluded that, if the stimuli are mild, only the first phase occurs; if the stimuli are greater the second phase also occurs. The adrenalin thus liberated acts through the blood on the iris muscle, effecting dilatation of the pupil. The dilated pupil crowds the iris against the trabeculae. Under great emotional stress large amounts of adrenalin are poured into the blood stream. As a result the iris is crowded more and more into the angle of a shallow anterior chamber, precipitating an acute attack of glaucoma.

Case reports

Case 1

I. R., a white woman 59 years of age, was operated for repair of cystocele and rectocele on October 22, 1953. Past history brought out that she had never had any previous surgery. She stated that she has never had any previous eye trouble. Two days after surgery at noon she became very nauseated and vomited profusely. She complained of sudden severe loss of vision. When seen about 30 minutes later, a diagnosis of acute severe bilateral closed-angle glaucoma was made.

The examination was rather difficult. The patient was in a state bordering on panic. Extreme pain was evident and she kept repeating, "Am I going blind. . . . ?"

Ocular examination. Both eyes were intensely injected. The corneas were steaming. Both pupils were widely dilated. Vision: O.D., counting fingers at four ft.; O.S., counting fingers at five ft.; no fundus reflex was present.

There was no need to use a tonometer as both eyes were stony hard to digital pressure which aggravated the pain. The patient was taken to the operating room as soon as it could be made ready. In the meantime two-percent pilocarine drops were dropped into both eyes every two minutes. The tension remained unaltered.

Operation. In the right eye, it was necessary to make a scratch incision because the anterior chamber was extremely shallow. Immediately on opening the anterior chamber the iris bulged out into the incision and a broad base iridectomy was performed. The left eye was treated similarly except that the tension was slightly lower and the iris did not bulge.

Her postoperative course was uneventful. The pain subsided almost immediately; the complaints did not. The tension next day was below normal and at no time in her follow-up course, to February 5, 1957, did the ocular tension rise again.

Follow-up. Within two months her vision returned to near normal. On November 30, 1953, her vision was: O.D., 20/100; O.S., 20/100. It continued to improve gradually and on December 28th it was: O.D., 20/30; O.S., 20/30 with correction.

Early in 1956 lens changes in both eyes began to interfere with vision and on February 5, 1957, vision was: O.D., 20/70; O.S., 20/70 with correction.

It is felt that eventually it will be necessary to remove her lenses for better vision and to prevent any further episodes of acute closed-angle glaucoma.

Personality analysis. This patient was under observation for four years and she is still seen from time to time. She is always in a state of anxiety, flitting from one subject to another. Her sentences are never completed and at no time does she listen to anything told her. Her visits consist of an examination plus a 15-minute recitation, well documented, of all her various ills. It was almost impossible to stop her. The recitation began with her fear of ensuing blindness and left out no detail concerning her abdominal, gynecology, and many other complaints. Her meek, docile husband sat by and nodded agreeably with the mention of each symptom.

Even when it was demonstrated to her that she had 20/30 vision in each eye, she continued to talk of her blindness. It was useless to point out to her that she was able to perform all her necessary household duties and that furthermore she could read the paper with facility. After she apparently ran out of current complaints, she wanted to know again why she was going blind.

Her internist and gynecologist have had the same type of experience with her. We compared notes and found that she was quite thorough, never leaving out the smallest detail. Actually, she had them both convinced that she was going blind and they have inquired several times if it were so.

Comment. This patient had the basic anatomic structure predisposing to acute glaucoma—an extremely narrow, shallow anterior chamber. At surgery this was well demonstrated. She exhibited a great deal of insecurity mingled with an overwhelming anxiety. One can easily understand her anxiety concerning her vision. However, when her vision was corrected almost to normal the degree of fear did not abate. She maintained a good adequate home and apparently kept her husband happy to a degree. To some degree her complaints have some logic since her vision is slowly deteriorating due to development of cataracts in both eyes. Her gradual loss of vision may make her feel that she may have another episode like her initial attack.

When she spoke of the possibilities of surgery for her cataract, she wanted to have it done immediately. Perhaps she needs her many illnesses as a crutch for support. To kick her crutch out from under her would be unwise.

Case 2

M. T., a 64-year-old white man, was apparently in good health until March 6, 1953, when he was operated on when a gastro-jejunostomy and vagotomy was performed.

Past history showed temporary paralysis of right upper extremity one year ago. He also had some difficulty in speaking clearly at the time. There was complete recovery of function within a month; no organic lesion could be demonstrated at the time.

Three days after abdominal surgery he began to have severe pain in the right eye accompanied by severe loss of vision. At the time of examination the patient had acute congestive closed-angle glaucoma. The cornea was steaming, the pupil very widely dilated, the anterior chamber extremely shallow. The lens showed moderately advanced changes and the fundus could not be seen through the edematous cornea.

Tension to fingers in the right eye was stony hard in comparison to the left eye. (It is neither wise nor necessary to use a tonometer to evaluate the tension in such cases. Actually, it is dangerous to place the metal footplate of the tonometer on an eye so impaired.)

Surgery. The patient was taken immediately to the operating room and a broad-base iridectomy was done. The anterior chamber was extremely shallow and it was somewhat difficult to enter the eye with a thinned out four-mm. keratome. The iris bulged out immediately. The postoperative course was uncomplicated and tension remained quite good. The pain subsided also.

The patient was readmitted on February 21, 1956, for removal of cataract because of progressively poor vision. During all this time the tension remained normal. On September 22, 1956, the patient was admitted for a serpiginous ulcer with hypopyon. He recovered from this quite well but he is checked periodically.

Personality analysis. The patient was tremendously insecure. He had had a small restaurant for the last 30 years. Within the last five years he no longer was the breadwinner in the family, being dependent on his son for financial aid. He hated his set-up and spoke of it with a venomous attitude.

This attitude was superimposed over many anxieties. Anxiety about his financial status, anxiety about his vision, anxiety about the future, anxiety about his gastro-intestinal pains. The complete paralysis of right upper extremity one year before his first admission could have been an hysterical episode.

As a patient he never stopped complaining of his many ills, but concentrated on his eye. No amount of reassurance could stem his vociferous anxieties. At one visit he was told that his right eye was good, that he could well get around and do what he wanted with his normal left eye. After that, he went into a depression; he badly needed his ailment for a crutch which could not be taken away without doing him further harm. It was gently hinted once that he need not come to the office except every six weeks. He almost panicked and wanted to be seen every two weeks. So it was agreed. He continues his tirade of woes for a full 10 minutes at every visit. His anxieties are mounting and no amount of reassurance reaches him.

CASE 3

C. S., a 49-year-old white man, was seen first on April 16, 1948. He gave us a rather peculiar history, stating that on March 12, he had a sudden blurring of the left eye with severe pain. He was operated on in a doctor's office for glaucoma and had relief for two days, later he was hospitalized and operated on in bed.

The history in itself is hard to understand. Apparently a paracentesis was done each time. At time of first examination in our office his vision was: O.D., 20/40; O.S., counting fingers at three ft. Tension was O.D., 40 mm. Hg; O.S., 90+ mm. Hg. The left eye had an acute closed-angle glaucoma, the cornea very dull and cloudy. There was a wound below at the 5-o'clock position, probably the area of the paracentesis. The pupil was widely dilated but complete. There was no evidence of a broad-base iridectomy. No fundus reflex could be seen. The patient was extremely excitable.

He was operated on the same day as an emergency and a broad-base iridectomy was done. The anterior chamber was extremely shallow and a scratch incision was necessary. The iris popped out into the opening immediately.

When glaucoma was first diagnosed, the right eye was under treatment with pilocarpine (two percent five times a day). In spite of adequate treatment, including sedation, eight days later his right eye had a similar acute attack. Again he was taken to the operating room and a broad-base iridectomy was performed. He was operated on within 20 minutes after the attack and has maintained 20/30 vision in this eye and no further loss of field.

On May 26, 1948, he had another acute closed-angle attack in the left eye. It was felt that this was due to the large swollen intumescent lens which was again blocking the drainage angle. The lens was removed and the tension has remained good since then.

Personality analysis. Of the three patients he was by far the most excitable. He was unable to sit still in the office. When he entered the waiting room he paced up and down for almost five minutes, and then walked in regardless of the situation. He was a "bundle of nerves" and was in high pitch all the time. He never had a relaxed moment. He suffered from insomnia but burned energy every waking moment.

The patient was married to an attractive young woman who owned and operated a very successful business. A job had been created for the patient and evidently he was always interfering with the smooth running of the business. He was always in "hot water" and was doing his best to ruin a successful business. He almost succeeded.

There was no predicting what the patient would do. At times he would drive his wife's car with his limited vision, at 80 to 90 miles an hour. He drove it with reckless abandon but managed to avoid any accidents. He was maladjusted to people about him and to his environment. Actually he lived in a world of his own. Without being asked the patient would gladly tell everyone how the world and its people should conduct themselves. He talked continuously, compulsively, jumping from one subject to another. At times he could be quite humorous and entertaining. Five years after his initial surgery he died suddenly in his sleep with no cause that could be demonstrated. Perhaps he just burned himself out at the age of 54 years.

DISCUSSION

All three of these cases had several things in common. They all followed major surgery which precipitated severe emotional upset, and the iris was bombarded (with incessant) sympathetic stimuli plus the large amounts of adrenalin which had been poured into its blood stream. These two factors caused the shallow angle to be crowded even further,

resulting in full-blown closed-angle glaucoma.

The three patients were unstable, emotionally immature, full of deep-seated anxieties (fancied or real), nervous and tormented. They belong in a separate personality group, just as this type of closed-angle glaucoma belongs in a group separated from other types of glaucoma.

Summary

Conclusions

Three cases of emotional type of glaucoma are reported. The term itself is open to controversy and only time will tell if we can safely group this type of acute closed-angle glaucoma under this clinical entity. The predisposing anatomic and physiologic factors are reviewed and the role of the vitreous is stressed.

The pupil, which is influenced by acute emotional upsets as well as the adrenalin liberated at the same time, plays a major role. When the pupil becomes so dilated, there is a true mechanical block of aqueous drainage and a vicious sudden episode of acute closed-angle glaucoma is initiated.

1326 Maccabees Building (2).

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EARLY HISTORY OF CATARACT AND THE ANCIENT OPERATION FOR CATARACT*

Aryeh Feigenbaum, M.D.
Jerusalem, Israel

> Unravelling of the earliest history of couching for cataract is one of the most important pursuits in the history of ophthalmology.
> Julius Hirschberg.

In two previous papers[1,2] I tried to prove that India was the birthplace of the ancient operation for cataract and that the knowledge about the procedure must have spread from there to the Eastern Mediterranean in antiquity during or after Alexander the Great's Indian expedition. In the present study, after an indispensable recapitulation of some former material, additional evidence will be produced to this effect. As during the following several centuries, the Hellenistic and the Byzantine periods, until the eighth century C.E., a decline—inexplicable at first—has set in with regard to recognition of the procedure and its results, this fact will also have to be discussed, together with the factors de-

* From the Rothschild-Hadassah University Hospital and Hebrew University-Hadassah Medical School. In condensed form this study was first read at a special meeting of the Greek Ophthalmological Society in Athens, October, 1958.

termining the progress made during the Arabian period. Eventually, some later developments, until the era inaugurated by Daviel, will be touched upon.

1. Evidence for India being the subcontinent from which the needling operation originated

The first mention and description of cataract operation in the western literature appears—as if all of a sudden—in the encyclopedic work of the Roman writer Celsus De Medicina (about 30 A.D.). This is the first evidence for the method known in the Hellenistic world. Celsus must have taken it over from the Alexandrian school.

History of cataract operation, a most fascinating subject, was until the time of Jacques Daviel (1748), who invented the method of extraction, the record of couching (or needling) for cataract. This was a method of displacing the opaque crystalline lens from the pupillary area and depressing it, more or less violently, into the vitreous by a kind of paracentesis (or nyxis) of which in olden times the route of approach was the posterior, the so-called scleronyxis (fig. 10-B, posterior operation).

The anterior approach, by keratonyxis (fig. 10-A, anterior operation) is a much later invention, first used in Europe at the beginning of the 19th century; while in India both methods were (and probably still are) used by quacks (Elliot[3]).

Celsus'[4] description (Spencer's translation) reads:

> Now either from disease or from a blow, a humour forms underneath the two tunics in what I stated to be an empty space; and this as it gradually hardens is an obstacle to the visual power within. And there are several species of this lesion; some curable, some which do not admit of treatment. . . . Generally too the case is worse when the suffusion (cataract) has arisen from a severe disease, from severe pains in the head or from a blow of a violent kind. Old age is not favourable for treatment . . . , neither is childhood favorable, but rather intermediate ages. . . . And in the suffusion itself, there is a certain development. Therefore we must wait until it is no longer fluid, but appears to have coalesced to some sort of hardness. Before treatment the patient should. . . . Then he is to be seated opposite the surgeon in a light room, facing the light, while the surgeon sits on a slightly higher seat; the assistant from behind holds the head so that the patient does not move; for vision can be destroyed permanently by a slight movement. In order also that the eye to be treated may be held more still, wool is put over the opposite eye and bandaged on; further the left eye should be operated upon with the right hand, and the right eye with the left hand. Thereupon a needle is to be taken pointed enough to

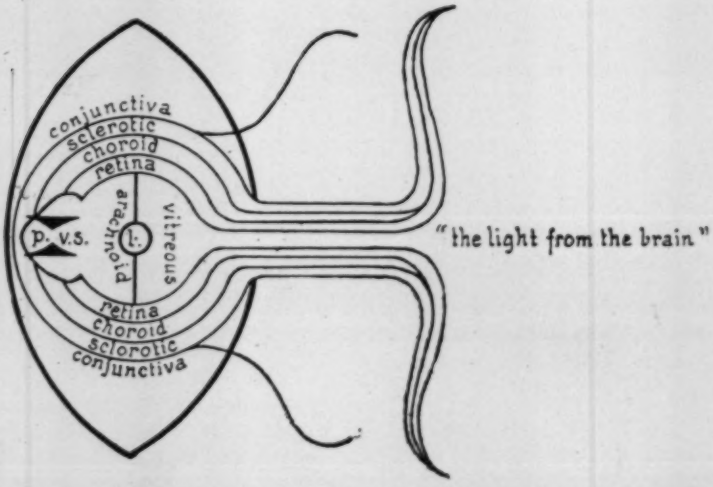


Fig. 1 (Feigenbaum). Structure of the eye, from the textbook of Hunain ibn Ishaq (809-877). Modelled possibly, according to Meyerhof, after a lost Greek original. (l) Lens, (p) pupil, (v.s.) visual spirit.

penetrate, yet not too fine; and this is to be inserted straight through the two outer tunics at a spot intermediate between the pupil of the eye and the angle (of the palpebral fissure) adjacent to the temple, away from the middle of the suffusion, in such a way that no vein is wounded. The needle should not be, however, entered timidly, for it passes into the empty space; and when this is reached even a man of moderate experience cannot be mistaken, for there is then no resistance to pressure. When the spot is reached, the needle is to be sloped against the suffusion and should gently rotate there and little by little guide it below the region of the pupil; when the suffusion has passed below the pupil it is pressed upon more firmly in order that it may settle below. If it sticks there the cure is accomplished; if it returns to some extent, it is to be cut up with the same needle and separated into several pieces, which can be more easily stowed away singly, and form smaller obstacles to vision. After this the needle is drawn straight out; and soft wool soaked in white of egg is to be put on, and above this something to check inflammation; and then bandages. Subsequently the patient must have rest, abstinence, and inunction with soothing medicaments. . . .

This description of the procedure of needling, from which inessential directions have been left out, is a fairly good one but obviously second hand since Celsus himself was no medical men. *Suffusio*—for which the term "cataracta" was substituted as late as in the 11th century—is a translation of the Greek *hypochyma* (or hypochysis) and means the appearance of some solidified matter behind the pupillary area; in an "empty space" means the space behind the iris or between the iris and the lens, and not involving the latter.

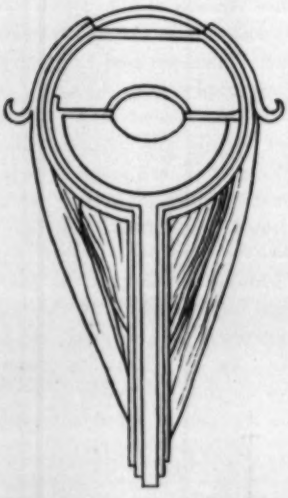


Fig. 2 (Feigenbaum). Schematic eye, from *De fabrica corporis humani* of Andreas Vesalius (1514-1564).

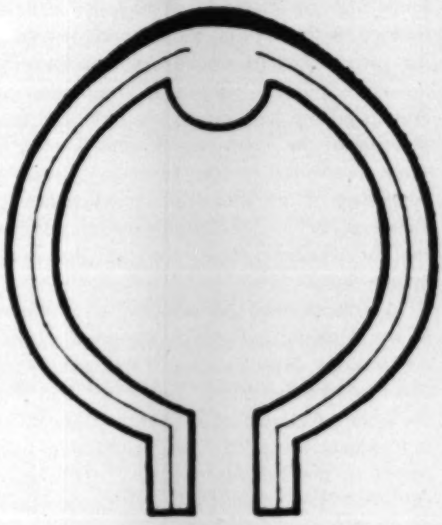


Fig. 3 (Feigenbaum). Sketch from *De oculo* of Fabricius ab Aquapendente (1537-1619), showing correct position of the lens within the eyeball.

Ignorance of what was really done in dislocating the cataractous lens was due to an almost complete ignorance of the anatomy of the eye, especially of the correct position within the eyeball of the crystalline lens. The latter was regarded, until the 17th century, and mainly due to the authority of Galen, the central organ of vision, and was accordingly placed in the center of the eyeball, as shown by the first known diagram of the eye (fig. 1) in the ninth-century textbook of Hunain ibn Ishaq[5] (Meyerhof's translation) which was uncritically copied even by the great Vesalius in the 16th century (fig. 2). For the coucher it was, therefore, unthinkable that the lens should be attacked—and frequently even injured—in an operation, the aim of which was to restore vision. And this discrepancy between the very limited theoretic

knowledge of anatomy, physiology, and pathology of the eye and the practice of couching for cataract continued, for reasons to be mentioned later (although Fabricius ab Aquapendente had shown, in 1600, the true position of the lens* [fig. 3] and Kepler, in 1604, discovered its true function), until the beginning of the 18th century, when Michel Brisseau (1705) definitely proved by autopsy that the dislocated cataract was the dislocated opaque lens.

In considering the possible forerunners of the Alexandrian school, the great ancient civilizations, Mesopotamia, Egypt and Persia can readily be excluded. In ancient Babylon, the Code of Hammurabi (before 2000 B.C.) is frequently quoted[6,7] as mentioning payments to the surgeon for the "needling of cataract" or forfeits if he were unsuccessful in this operation. This, however, is founded solely on an erroneous interpretation of the word *nagabti* or *naqabtu*, which most probably means a lacrimal fistula (Hirschberg[8]); in semitic languages the root *naqab* denotes a "hole."†

In ancient Egypt no indication can be found in the medical papyri of the knowledge of cataract or cataract operation, though this, through a misunderstanding of the expression "uprising water," has been claimed by some authors (Magnus[10]), but has been invalidated by Egyptologists and ophthalmologists alike.[8,11,12] Old-Persian (Mazdayan) medicine as presented in Zend-Avesta is altogether unprofitable. On the other hand, there are weighty proofs that ancient India most probably was the birthplace of the method (the same as, by the way, of skin grafting and rhinoplasty).

"Suśruta (generally placed about 1000 B.C.) was the first Ayurvedic surgeon to deal systematically and most exhaustively and elaborately with the development of the anatomic structure and the physiologic factors, the pathologic changes, the symptoms and the treatment of ocular diseases," says Bidyâdhar.[13] If we follow the (obviously free) translations by this and by another modern Indian ophthalmologist[14] of the paragraphs describing the needling operation, as taken from the voluminous ancient medical work entitled *Suśruta Samhita* and their (sometimes even a little overenthusiastic) comments, one necessarily, has to assume a period of several centuries of earlier experience—before Suśruta—leading by stages to this perfection.

Personally, I have to confess from reading the relevant paragraphs in the rigid English translation* of Suśruta Samhita,[15] that I was unable to decide at first whether *linganâśa*, explained as an obstruction or choking up of the pupil with "cataract," was identical or not with what we today call more or less complete opacity of the lens; this was probably due to my inability to penetrate into the utterly strange world of ideas expressed in terms extraneous to the Western mentality. Later I also perused the translation of these paragraphs into German by Prof. Weber (in which a number of English expressions, words and phrases, taken from T. A. Wise, 1867, are inserted for comparison, where the literal meaning seemed dubious), quoted by Magnus;[10] another German translation by Prof. G. Oppert, reproduced by Hirschberg, as well as the more recent translation into German (of the whole chapter concerning the surgical treatment of linganâśa) by A. Albert M. Esser.[16]

It appears that *linganâśa*—for "cataract"—carries the idea of some corrupt humor, a

* According to Morgagni (1682-1771) Fabricius succeeded in this after having cut a frozen eyeball (using ice and salt).

† As to etymologic derivation, I am at variance with Hirschberg (nagb = chasm, source). Cautery of the lacrimal abscess was the treatment of choice in ancient times and was warmly advocated by Desmarres even in the middle of the 19th century. Cautery in the close vicinity of the eye might, through the intense heat, cause loss of vision when carelessly executed.[9]

* Chapters I-XIX (pp. 1-105) deal with ocular diseases; chapter VII discusses the pathology of the disease of the pupil of the eye; and chapter XVIII the "Treatment of the disease of pupil and crystalline lens," including the "Surgical treatment of Kaphaja Linganâśa."

kind of phlegm developing behind the pupil, similar to the Greek conception of *hypochyma* (inspissated humor) or *glaucoma* (grayish-blue discoloration of the pupil). The latter term was taken over at a much later period as *ma zarka* (blue water) by the Arabs with whom the notion of an inspissated humour descended (as a fluid) from the brain (*nuzul el-ma*) was connected.

Moreover, a comparison of these various translations, made independently, is of the utmost value for getting closer to the meaning of the original text. A still better understanding of the latter can be obtained by putting the ancient text in juxtaposition with later observations on the traditional way of couching in India, as well as with a comparison to the classical Arabian procedure during the 10th and 11th centuries.

Here is a literal translation (into English) of the passages rendered into German by Esser comprising p. 452, verses 19-34, and p. 453, verses 1-7 of the Sanskrit edition used by him. Verses 19-34, page 452, read:

In linganâśa arising from the phlegm I will now review the procedure (leading) to cure
Unless the corrupt basic humor at the pupil has the shape of a half-moon, a drop of sweat and a pearl, and appears solid
Or mobile, thin in the middle or striated, intensely shining,
Painful or entirely red.
When the sagacious (physician), at a time not to warm or cold, in the one (treated) with fatty drugs or sweating remedies,
The fastened (patient), who sat down and looks straight at his own nose
From the outer corner both white parts separated from the black
And both eyes, that are free from sirajala, has opened completely,
May the physician, on both sides neither below nor above, at the hole made by nature (pupil)
With a lancet, which has a barley-shaped point,
And which is kept with forefinger, middle finger and thumb in his hand
With the right hand the left and with the left hand the other (eye) perforate carefully and assuredly.
Hereby simultaneously the appearance of a drop of water as well as a noise would take place at the perforation.
The experienced (physician) after having sprinkled with woman's milk what was perforated just now,
He should, whether the corrupt basic humor is solid or even mobile, let the eye from the outside,
After having well fixed the lancet, foment with broken off parts (twigs) that expel wind.

Verses 1-7 on page 453 read:

Thereupon he should scratch the pupil with the point of the lancet.
Shutting the side of the nose, which for the surgeon is on the other side of the eye,
The phlegm arisen at the pupil must be removed through drawing up into the nose.
When the pupil shines like the sun without clouds,
Then it (the pupil) has to be considered well scratched, also the one which is free from pain.
When shapes are seen, he should then gently remove the lancet,
And after having anointed the eye with melted-down butter, he should cover it with a strip of fabric.

One has to admit, this translation of the verses of Suśruta pertaining to the operation for cataract does not present us with clear details and does not exactly mark their sequence. The advice of Magnus will, therefore, be followed and this text will be compared with a full description of the (modern) couching operation, as performed some 134 years ago in India in the traditional way by a native oculist. This has one great advantage over the needling operation performed clandestinely today by the Indian quack (which, by the way, in spite of its low-grade execution, still follows the main lines of ancient tradition) as it obtains from a period when the method of couching was not yet exposed to odium in the East and still had its legitimate exponents in Western Europe. Here is a verbatim translation from the German* of the report of an eye witness, Dr. Breton of Calcutta, referred to by Magnus:

The Indian oculist uses for cataract operation two instruments, the lancet for perforating of the outer tunic of the globe and the *sulaee* (the needle) for depression of the cataract. Around the upper part of the lancet, 1/10 of an inch† from its point, a thread is wound, whereby a deeper thrust into the eye, than the point marked, is to be avoided. The

* It is most probably a re-translation into English (Magnus").

† In the text it reads "1/10 of a line" which is an obvious error (one line = 1/12 of an inch = some two mm.).

depression needle is of copper and of a cylindrical shape; it has the thickness of the feather of a crow, is some 5 inches long and, beginning from its posterior end it is tapering off until half an inch before its pointed end. The latter part of the instrument, half an inch long, has the shape of a three-sided pyramid and below the pyramid there is a short neck, the thinnest part of the instrument. One inch from the point, a thread is wound around the needle, in order it should not enter further into the globe."

After having described the instruments, Dr. Breton gives the following description of the operation which he had witnessed repeatedly:

After preparing the instruments, the Mohammedan oculist Sautcouree, who performed the operation in the presence of Mr. W. Twining, a native medical student and myself, on 3rd July, 1824, seated the patient on the floor, after having previously bandaged the healthy eye, in order to prevent movements of both eyes as much as possible. Behind the patient an assistant was placed who, supporting the patient's head on his thighs, firmly held it with both hands, for the sake of immobilization. The surgeon then sat down close to the patient on a *morha*, a stool one foot high, and, after having put the hands of the latter into his girdle, bowed three times, invoking the help of the Almighty for a favourable result, and started the operation on the left eye. Lifting with his left thumb the upper eyelid (a retractor is never used), he put his fingers on the forehead of the patient and, telling him to look in the direction of his nose, he presently punctured the globe with the lancet he held in his right hand. The puncture was done through the sclerotic, at a distance of one tenth of an inch from the corneal border and somewhat below the axis (horizontal diameter) of the pupil, and the lancet, which had been thrust into the globe up to the thread (some 2.5 mm.), was taken out. The opening was big enough for introducing the needle (called *sulaee* —gross needle*—by Sautcouree) easily and carefully into the vitreous up to its neck (12.5 mm.); it then remained for half a minute suspended within the eye like a pendulum, supported by a piece of linen or cotton-wool, that was lying on the cheek, while the exact closeness of the sclera around the neck of the needle prevented it from falling out entirely. As long as the instrument remained still, there was no irritation. The eyelids were meanwhile closed and the patient was kept as quiet as possible. After half a minute the eyelids were opened with the fingers of the left hand, the point of the needle, held parallel to the axis of the pupil, was led to the upper and outer part of the lens and the latter was softly depressed downwards into the vitreous, where it was kept for some seconds. Thereupon the point of the needle was carefully removed from the lens, and as often as the latter surged again, it was depressed until it did not appear anymore. . . . The eyelids were then closed, the needle remained for some seconds suspended in the eye as before, and the patient had to keep quiet altogether. Meanwhile a red-hot ball made from artichokes and clay, lying on a flat earthen vessel, was held near the eye for fomentation. . . . Then the lids were opened again and the patient had to eject air through the nose several times, and Sautcouree gave him two or three gentle knocks on the head. . . . When no opacity was visible behind the pupil the patient was asked, whether he was able to distinguish objects. . . . When he could do this, the operation was considered finished, the needle was removed, a pad of cotton wool was put on the eye and the latter bandaged. After an hour or two the patient could go home and was told to stay at home for some days without touching the bandage. . . .

* Of which the opposite seems to be *śalākā*—fine instrument (Esser).

Magnus[10] adds a series of similar descriptions from other parts of India, which show some slight variations in the shape of the instruments or in the execution of the operation, without differing fundamentally from the one quoted, and preserving everywhere the peculiar features of the ancient Indian operation. Hirschberg,[8] while quoting Major H. E. Drake-Brockman, I.M.S., and his description of some instruments for the Indian needling operation at the end of the 19th century (fig. 4) observes: "Together with the ancient procedures of cataract operation also the ancient instruments have been preserved in the East until our own days."

Comparing the ancient and modern Indian method, it is remarkable indeed how they resemble each other in all their main features. Both were performed with two instruments in a number of steps (Magnus). True, in the ancient text the break between the first step of perforation (verse 32: "The experienced [physician] after having sprinkled with woman's milk what was perforated just now") and the next one (verse 33) is not marked too well, but it is recognizable (conceded also by Hirschberg.[17]) In both descriptions the eye operated upon is treated with fomentations during the operation, and at the end the peculiar practice is described of blowing the nose or pulling in air through the nose.

India, with its great frequency of cataract

and its early maturation (today around the age of 40 years), conditions probably existing there for millennia (and necessity being the mother of invention), seems a priori to have been a fertile ground for the evolvement of the needling operation and its development, once it was invented. At the time of Alexander, the procedure must have been already of a venerable age in that vast country, and the art of couching cultivated by well-trained specialists.

When trying to determine the time when the Indian method might have been transplanted into Greek medicine, we have to remember that in the Hippocratic writings there is no mention of a cure for *glaucosis* or *glaucoma*, terms already mentioned, which undoubtedly served in the fifth and fourth centuries B.C. for indicating "cataract" of every description.* So the limits of the presumptive "mute"† period can be roughly from 300 B.C. and 30 A.D., comprising the earlier and later Alexandrian period of which we know so little, as all the medical writings of this famous Alexandrian school were lost and, indeed, not a single special work on eye diseases in Greek has survived to our times. Whatever has come down to us of the views and practices of the ancients in this field is dispersed in general medical works or quoted in fragments by later authors (Galen, Oribasius, Aëtius, Paulus and Rhazes).

Hypochyma of the Alexandrians (third and second centuries B.C.) was more or less representing the operable variety of cataract, while the older term *glaucoma* was probably reserved by them for the incurable variety.

As to the question whether the method of couching for cataract may not have come up independently in different places of the ancient world, the following may be said: True, some general ideas about elements, life, health, illness (exactly as the hypothetical conception of some corrupt matter, appearing in diseased tissues, be it linganâśa, hypochyma, nuzul el-ma, cataracta, as mentioned previously and so forth) may have originated independently in different and far away countries and look identical, but this will frequently be due to the convergent working of the human mind in general.

Thus, apart from having to prove the early antiquity of the method of couching for cataract in India (or making it very probable) there remains the following gap to be filled in: How did the knowledge about the procedure travel from India to the Eastern parts of the Mediterranean? There seems, however, to be no question about the role played

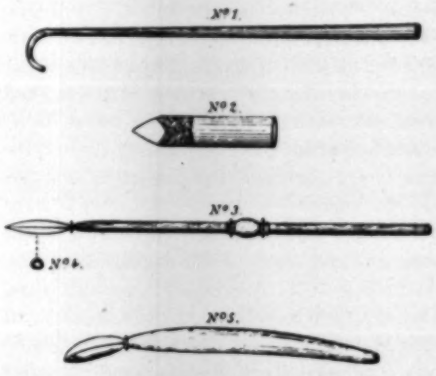


Fig. 4 (Feigenbaum). Surgical equipment for the Indian needling operation, as depicted and described by H. E. Drake-Brockman at the end of the 19th century: (1) Iron lid elevator; (2) lancet-shaped knife, 4.5 cm. long, 7.0 mm. from its tip a cotton thread is wound around the instrument (to prevent deeper penetration); (3) copper couching needle, 9.5 cm.-long handle, separated at its neck by a narrow ring from the needle proper, which is 1.6 cm. long and 3.0 mm. wide at its largest diameter, and (4) three-edged in cross-section, with clearly rounded-off edges. (5) Another copper couching needle (Punjab model). Needle proper 1.8 cm. long, 5.0 mm. at its largest diameter, joining the handle under obtuse angle.

* By no means did these terms serve then (and many centuries afterward) for what we today would call glaucoma.[84]

† Not quite mute. From Sudhoff (1924) who based his work on a finding of the classical philologist, K. Kalbfleisch, Sasse (Antike Kenntnisse. Ciba-Zeitschrift, **8**: 3079 [No. 93] 1959) quotes a passage from Chrysippus, one of the most prominent representatives of the Early Stoa, in which needling of cataract, apt to restore sight, is mentioned incidentally in a dialectic disputation. The passage was written in the second half of the third century B.C. and at least 80 years after Alexander.

especially by Alexander's expedition at the end of the fourth century in opening up traffic in both directions, either by land (via Persia) or by sea (to and from the Red Sea). We have to remember that Alexander of Macedonia, a true disciple of Aristotle, the great Stagyrite, took along with his expeditionary force a number of men of science, among them physicians, whose duty it was to collect material and gather knowledge which came their way during the march towards the "Eastern border" of the *oikumene*, "the inhabited world."

On the other hand there are the testimonies for the excellence of the Indian physicians at that period by Megasthenes who under Seleucos Nikator (321-281 B.C.) came as ambassador to the enlightened emperor Chandragupta[18] and in his *Indika* (of which only fragments exist) reports "that in India the physicians have to care even for the foreigners and that next to the foresters, the physicians belong to the most learned people."[19] Arrian (about 160 C.E.) in his *Indika* tells us that "the Greeks had no antidote for their bite (that is, the bite of snakes) but the Indians are provided with one; and some that were possessed of the secret Alexander had always in attendance. . . . The Indians have remedies also for other diseases . . ."[19]

Now the date previously mentioned of the time Suśruta lived, is admittedly uncertain. According to T. A. Wise[20,21] Suśruta (and Charaka) are mentioned already in the great epic Mahábhárata of which the first version was concluded about 400 B.C. and the latest —shortly after the reign of the renowned king Aśoka (250 B.C.). Furthermore, A. F. Hoernlé (1907) quotes the Hindu tradition placing the earliest Hindu medical schools of Atreya and Suśruta at some time in the sixth century B.C., a date supported by the Vedas (Sarton[22]). As the climate of India is most unfavorable to the preservation of documents written on such perishable materials as parchment, bark or leaves, there is a complete lack of ancient manuscripts, and a fragmentary Buddhist religious text of the fifth century A.D. is the earliest one found so far.[23] But it is remarkable that the very first inscriptions engraved on rocks and pillars between Peshawar (north-west) and Orissa (east), the second of the 14 commands of the famous king Aśoka, already contain very enlightened instruction about regular medical care for man and animal, from which it follows that at that time (third century B.C.) a highly developed system of medicine must have existed in India.

In the plain around Peshawar, called Gandhâra in ancient times (on the northwestern Indian border), a grand style of Graeco-Buddhistic art developed from the period of Alexander onwards of which a recent exhibition in Paris[24] gave eloquent evidence. Recently also (1958) some Greek inscriptions with an Aramaic translation, hewn on rocks and dating from the third century B.C. were found by archeologists in Afghanistan (near the border of contemporary Pakistan), strengthening the view of a closer liaison between India and the Hellenistic world.

2. Stagnation during the Hellenistic period and part of the Byzantine period

It is rather strange to find what considerable time it has taken until the method of couching for cataract, newly imported into the Eastern Mediterranean, has become established as a more or less accepted procedure, and it is not easy to understand why the latter has for centuries remained in a state of prolonged infancy. In this connection it is almost pathetic to quote Hirschberg's[8] concluding remarks on the last page of the first volume of his *History of Ophthalmology*, dealing with antiquity. In referring to a comparison of the all but two treatises on eye surgery extant from antiquity, that of Celsus and that of Paulus of Aegina (seventh century), he states: "In the domain of ophthalmic surgery—we have to acknowledge it candidly with sadness—progress is insignificant during all these six centuries; we might

say, eight centuries, if the writings of the Alexandrians were in our possession." And in no other chapter of eye surgery does this statement hold truer than in the important and central chapter of the surgical treatment of blindness from cataract.

When calling to witness authors after Celsus, from the first century on, we find here and there good observations on cataract but, on the whole, little experience and small confidence in the operation for cataract. This may be illustrated by the following:

Demosthenes Philalethes (first century) was one of the most distinguished representatives of the later Herophilian school*; his book on eyes (Ophthalmikós or Perí ophthalmōn) obviously was the main source of all later writers on the subject. He is quoted by Aëtius of Amida (sixth century) in a chapter "on hypochysis" (Hirschberg,[25]) which contains some remarkable sentences about the early signs of cataract† and its classification, but seems to reveal diagnostic and therapeutic embarrassment. After enumerating the various appearances of the pupil in hypochysis he concludes: ". . . but others become grayish-blue, and these are incurable"—a remark which Hirschberg finds somewhat strange.

Rufus of Ephesus. The vague anatomic conception coupled with the uncertainty of which kind of cataract would offer the best chance for a needling operation, is also reflected in the famous dictum of Rufus of Ephesus (first and second centuries A.D.): "Glaucoma and hypochyma were believed by the ancient authors to be one and the same disease, while later authors declared glaucomata to be a disease of the crystalloid humor (the lens) which through drying-up changed into bluish-gray; hypochomata on the other hand to be a pouring forth of fluids which become inspissated between the iris and the crystalloid. All glaucomata are incurable, the hypochymata are curable but not all of them."

The differentiation between "glaucoma," a form of incurable blindness, from "hypochyma," sometimes curable (by implication—by means of the needling operation) seems to be based more or less on the final outcome of the operation.

Galen. The confusion does not clear up even with Galen (second century), the greatest figure in Greek medicine after Hippocrates, who in his commentary to the latter's aphorism surprisingly (and in contradiction to Rufus) declares that "the term glaucosis includes the disease called hypochyma." But according to another statement made by him (in *De usu partium*) "glaucosis is a drying up and excessive congelation of the crystalloid humour, and leads to blindness."

It thus appears that Galen was not entirely convinced of the curability of hypochyma by operation, in spite of some remarks extant from his lost treatises on eye diseases which might point to the contrary. Though practising surgery on a large scale, especially during his first stay in Rome, it seems that he himself did not bestow great pains on the needling operation nor did he apparently have enough time for it.

Antyllus, an eminent Greek surgeon of the second or third century, seems to be an exception as far as confidence and experience are concerned. But what we know of his cataract operation is a fragment quoted in Arabic writings (Rhazes' *Continens*) of the ninth century which we have to accept with some reservation. We shall therefore have to return to this author later also in connection with the question whether the ancients knew anything about cataract extraction.

Oribasius. In his *Synopsis* Oribasius (fourth century) who has the great merit of having corrected many errors which have crept into the text of Galen's writings, quotes

* The prominent Alexandrian Herophilus, founder of human anatomy and first describer of the human eye, flourished in the third century B.C.; his treatise *Perí Ophthalmōn* is lost entirely.

† ". . . in the beginning of the hypochysis the following occurs to the patients. It seems to them as if minute flies and some darkish specks are incessantly floating before their eyes; others perceive woolly strings and hairy structures or cobwebs."

the already mentioned dictum of Rufus.

Aëtius of Amida. This distinguished writer of the sixth century is still unable to define the relation between glaucoma and hypochyma. In the chapter "peri glaukoseos" he says that the second kind of glaucosis which "originates from preceding hypochyma" (it could be intumescent cataract causing secondary glaucoma) "is incurable" —the same which, by the way, applies to the first kind. Aëtius seems to have small interest in surgery, does not discuss operation of cataract at all (which Hirschberg also finds a little strange), and in one instance only (in chapter XXX) he advises "to apply one of the drugs used in the needling operation."

Paulus of Aegina (seventh century), in his short but precise and careful presentation of the opthalmology of the ancients,* makes a clear diagnostic distinction between conditions of glaucoma and hypochyma (Rufus). In chapter 21 dealing with couching for cataract he declares: "All hypochymatous (cataractous) patients see light (have light perception) either more or less, thus we distinguish hypochymata from amaurosis and glaucoma, as those afflicted with amaurosis and glaucoma (that had run its course) do not see light (have no light perception) altogether." He then gives a description of the needling operation, on general lines identical with that of Celsus, but in greater detail and clarity, as befits an experienced specialist. True, he quotes Galen here and there, but one has the impression that this is only paying lip service to the great teacher and not that he had learned anything from him about the needling operation. Paulus, living in the Byzantine period, seems to be one of the first Greeks who seriously and intensively cultivated the procedure. In this respect he is the forerunner of and example for the renowned Arabian eye specialists of the 9th to 12th centuries by whom he is quoted, often literally.

* Reconstructed and edited by Hirschberg[8] as the "Greek Canon of Ophthalmology."

In spite of the rounded-off teachings of Paulus of Aegina, we are astonished to find it confirmed, on the whole, that during the 900 years or so, that lie between him and the introduction of the procedure into Alexandria, the needling operation had made no progress in Greek surgery until the following centuries, in the so-called Arabian period.

Apart from some possible contributory factors of a more general nature (political, socio-economic circumstances,* and so forth) which are beyond my competence, and are therefore better left out from this analysis, there were in my opinion two main reasons for this stagnation, each dependent upon the other, so that it is difficult to say which of the two might have been the primary one.

It would appear that in the first instance it was the deficient technique of the Alexandrians, employed before Celsus and during the subsequent centuries. A technique inferior to the one of the more experienced Indians must have in the second instance, led to so many failures that its use was discouraged. Thus neither devotion to the method nor competence in its execution could evolve.

3. The appearance on the ophthalmologic scene of the Arabs (ninth century) and the importance of the instruments used in couching

The Arabic language, as it spread over the lands of Moslem conquest, was rapidly adopted by the educated portion of the population, whether Moslem, Christian or Jewish; all of them contributed in equal measure to the flowering of the Arabian scientific literature during the following centuries. In reviewing the authors of the medical literature written in Arabic, from the middle of the ninth until the beginning of the 13th century, we learn

* In contradistinction to the reign of the first two Ptolemies—from the reign of Ptolemy III on (middle of third century B.C.) until the conquest of Egypt by Caesar—disintegration, social, political, and cultural, had set in (Durant[28]).

about a class of scientifically well-equipped and experienced practitioners in our speciality who had a humane attitude toward the patient. Some of them had performed hundreds of needling operations. We learn about their dexterity, devotion, and—frequently enough—excellent results, which must have increased their confidence in their technique for the surgical treatment for cataract.*

In addition to their abilities (or maybe rather intimately connected with them) one main difference stands out in their procedure in comparison with that of their Greek predecessors, namely the use of the instruments (figs. 4 to 9, and 14). Therefore, this particular aspect of the practice of some of them will be reviewed.

HUNAIN IBN ISHAQ (809-877 A.D.), a distinguished oculist and scholar, the author of the oldest Arabic textbook on eye diseases founded on Greek tradition, was a Nestorian.† Hunain's book, 10 treatises on the eye, of which the original Arabic manuscript was found (two copies), edited and translated into English with commentaries by Meyerhof[8] in 1928, contains a remarkable paragraph on the surgical treatment for cataract. It should be quoted here in its essence for a number of reasons. It is based entirely on the procedure adopted by the Greeks (Celsus and Paulus), is still untouched by the renewed Suśrutian influence on Arabian cataract surgery and contains very interesting remarks on the dangers of couching, such as are not to be found in any of the earlier or later treatises on ophthalmology. Emphasizing this, Meyerhof thinks the chapter is original* and may have been extracted from a lost work of Galen, a probability to which I subscribe:

> . . . If you intend couching (operation), put your needle at the extremity of the eye in the outer corner, hold the eyeball with your fingers and let your (other) fingers be at the origin of the needle. Then pierce it through, taking care that the eyeball does not slip from under your finger, and the needle reach the layer of the cornea or enter between the conjunctiva and the cornea and so reach the black of the eye and tear it. For this would cause greater damage than its piercing, because it would make a hole in the conjunctival membrane by which the (bad) humours would flow into the eye. This would result in throbbing and severe pain which is not apt to heal easily. After you have pierced it (the eye), beware of turning your needle in the wrong direction and of reaching the back of the uvea from inside and tearing it, for that would destroy its pupil and it is (an injury) not likely to be cured. Deal gently with the cataract, until you have removed it gently from its place, and beware of treating it roughly, lest you should tear its layer, and the cataract be scattered, so that it would be difficult for you to collect it and bring it back. . . .

There follow instructions regarding dressing and bandaging the eye, as well as directions for the postoperative behavior of the patient:

> . . . Know that after three days have elapsed since his day (of operation) under your treatment without any damage occurring to the eye, it has escaped it, as most damage occurs in the course of the first three days. Sometimes severe headache and throbbing occur, and sometimes it (the eye) is lost and sometimes it recovers, but it is saved in only a few cases. In general its (the cataract's) treatment is a risky one and its benefit cannot entirely be relied on. There are damages which I cannot mention at all, as I have abridged my book. . . . I have spoken and explained to you about the treatment of cataract in order to enable you to treat it, if you like to undertake its treatment with full knowledge of its dangers (Meyerhof[8]).

This very outspoken view of an experienced man throws some light on what was

* The thorough translations from the Arabic by Hirschberg, Lippert and Mittwoch[27, 28] and their commentaries will give most useful information on this.

† This Christian sect whose followers, banished at the end of the fifth century from Byzantium on account of "heresy," settled partly in Mesopotamia. They are known for their growing activity during the next centuries in various branches of science and, especially, for their translation of Greek authors into Syriac and Arabic, thus saving from oblivion a great heritage and tradition. Jews also played a very important part as translators.

* It is marked as "correction" in one M.S.; such corrections are numerous. The M.S. dates from 1003 (Meyerhof[8]). From a lost (11th) treatise, found by the famous Arab biographer Ibn Abi Usaibi'a (13th century) "annexed to this book (the 10 treatises) in which Hunain discusses the operative treatment of eye diseases"[8] must have been taken a quotation by Khalifa (Hirschberg[28]) with Hunain's warnings, reminding us vividly of those contained in the correction already mentioned.

said previously about hesitation and diffidence on the part of the Greek oculists, which can be gathered from their sayings about the method. It also stands in marked contrast to some sentences of Celsus, the layman, who must have repeated what he knew from the professional man: "the needle should not be entered timidly . . . when this is reached even a man of moderate experience cannot be mistaken." And finally, this seems to witness the greater risk inherent in the procedure as taken over by the Alexandrians.

Abu Bekr Mohammed ar-Razi (*Rhazes*, 850-923), one of the greatest Arabic physicians, who has undoubtedly performed eye operations himself, presents in his enormous work, the *Continens*, what Hirschberg[8] calls (a translation into Arabic of) "the best Greek description of cataract operation we know, the one of Antyllos." In this description we find much more detail and mature experience than in the one of Paulus, as evident from a juxtaposition of both texts.[8] In view of Hirschberg's opinion: "I have examined in detail 1267 paragraphs of the second book (where the eye diseases are included) of the *Continens* and carefully compared the quotations from the Greek with the original texts, as far as they are extant, and what I found was, that with amazing frequency, the quotation taken from the Greek was welded together with an addition, which one would not have recognized as such. Well now, we do not possess Antyllos." However, it does not seem easy to decide to what extent the views of Antyllos were modified in the process of a translation of this kind. I think the text seems to describe the use of two instruments (unlike the Greek procedure but as was customary with the Arabs of the 10th century), an opinion which I share with Magnus,[10] while Hirschberg does not accept it.

Abu'l Hassan at-Tabari (flourished around 970 A.D.), speaking of the needling operation in his book of the Hippocratic treatments, says: ". . . the outer tunic is perforated by the lancet, then the needle is pushed in gently, this is best and safest"[8] (quoted also by Khalifa,[28]).

'Ali ibn 'Isa (Christian, flourished around 960 A.D., died in the beginning of the 11th century) writes: "Take note, the external tunic is sometimes flabby and the needle does not pass through, so prick it (first) with a lancet, which has a roundish point, and thereupon insert the needle".[27] It is also said about him in a quotation by Khalifa: "'Ali ibn 'Isa tells us only, that the point of the cataract needle is three-edged and not very sharp."[28]

'Ammar ibn 'Ali of Mosul, who wrote his treatise during the reign of the Egyptian Sultan Hakim (996-1020) always used the lancet first for puncturing the eyeball and then the needle "of which the pointed end should be three-edged, in order that one of the three edges (better: sides) should without hinderance fall on the cataract."[28] He rejected the stereotype rule of operating with the right hand on the left eye and with the left hand on the right eye. He furthermore invented an original method for radical removal of a (soft) cataract by suction and describes a *hollow needle* for this purpose; the needle "should be exactly as the solid one, but the hollow one is thicker. The hollowing-out should extend from the beginning to the end, throughout. The spot with which the cataract is sucked in is a hole in one of its three surfaces. Whoever is going to use this needle, requires an assistant skilled for this purpose."[28]

Zarrin-dast (Gold hand) said in a Persian textbook on eye diseases with the Arabic title *Nur el uyyoun* (composed about 1088 and modelled after 'Ali ibn 'Isa and 'Ammar) ". . . the needling operation is performed in three ways: (1) with the lancet (and the needle), (2) with the solid needle alone, (3) with the hollow needle, as done by the people of Iraq, the Hindus and the Greeks." This sequence—by an obvious error—does not conform with the one just mentioned.[8]

Khalifa al-Halabi a later Arab author (middle of the 13th century) sometimes used

two instruments, first a lancet and then the three-edged cataract needle: "This procedure is safer than perforating the outer tunic with the point of the cataract needle and it is safer also with regard to the operation."[28]

SALAH AD-DIN (end of the 13th century) describes the cataract needle: "The head should be three-edged, not sharp as regards the three-edgedness, in order that, in whatever manner one turns the needle, one of the three edges (sides) should fall on the cataract and depress it."[28] He also says "some people let the needle be preceded by a lancet, only that part of the lancet remains free which passes through the outer tunic. Its measure is two-thirds of a barley-corn (three mm.")[28]

A succinct review of the instruments used in couching must needs start from Suśruta. As clear as it is, however, that the Suśrutian technique demanded two instruments, they seem to have had the same names in the ancient text, otherwise Esser, in his careful translation of the *Uttara-Tantra*, would not have applied the same term, "lancet," to both needles. On page 454 of the text, from verse 4-10 (Esser[16]), there are enumerated the attributes of a needle not fitted for the purpose as well as the qualities of a suitable one, and there appears to be no doubt that what is meant is the depression* needle: ". . . Eight breadths of a thumb in length (16 cm.) wrapped round with a thread in the middle . . . at both ends of the shape of a bud; of copper, of iron, of gold would be a faultless lancet" (fig. 4).

According to Celsus one instrument only is employed—the *acus*, a pointed needle. This may have resembled the one found in Cos, a silver needle with a ball between the pointed end and the handle, depicted by Meyer-Steinwegg (fig. 5), which in Hirschberg's opinion[29] could have been an ancient cataract needle of the kind used by the Greeks (later models of such a needle in Figure 14-a,b,c).

The instruments of the Arabs, as far as they are depicted in old Arabic manuscripts, were (according to Hirschberg): a cataract needle used for depressing the cataract, which in the majority of cases had a three-edged point (*miqdah*, fig. 6-A,C,D, and E), either solid or hollow, or sometimes a roundish point (*mihatt*, fig. 6B, 7-a, 7-b hollow); and a small lancet (*mibda'*, fig. 8) or a small knife (*ismil*), called *albarid* (fig. 9-a,b,c) which served for perforating the outer coats in the preliminary act—according to the Indian pattern.

It is indicative of Hirschberg's judgment and sensitiveness that, pointing to the resemblance between the Arab instruments (figs. 6 to 9) and the Indian instruments (lancet and three-edged needles), depicted in Figure 4, that without drawing far-reaching conclusions, he observes: "The attentive reader will admit that these illustrations and descriptions are apt to explain the drawings and texts taken from Arabic manuscripts."

It thus becomes evident that the Arabs, who admittedly drew their medical knowledge mainly from Greek sources, must have become acquainted, through a source not yet clearly identified, with the Indian variety of couching for cataract (with two instruments), and adopted it. Here a quotation by Wise,[20] in 1845, deserves mention:

Fig. 5 (Feigenbaum). Silver couching needle found in the island of Cos.

* The use of this word under the circumstances, anticipating the knowledge of depressing the cataractous lens, is admittedly anachronistic.

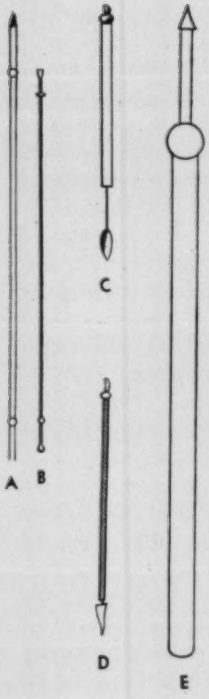


Fig. 6 (Feigenbaum). (A) Three-edged (hollow) needle (*miqdah*); (B) round needle (*mihatt*, lower end) added to manuscript of Ali ibn Isa; (C) (probably) three-edged needle (lower end), at the upper end a so-called "tail," used for marking the spot on conjunctiva where puncturing (with lancet or knife) was intended, from Abulqasim's *Surgery;* (D) (probably) three-edged needle (*miqdah*) with fluted handle, (E) three-edged hollow *miqdah*, depicted in manuscript of Salah ad-Din.

"Prof. Wilson, Calcutta, is of the opinion that the Arabians of the eighth century cultivated the Hindu works on medicine before those of the Greeks; and that the Charaka, the Suśruta, and the treatise called Nidhána, and so forth, were translated and studied by the Arabians in the days of Harun and Mansur (773 A.D.), either from the originals, or more probably from translations made at a still earlier period into the language of Persia."* Thus, there is reason to believe that the Arabic translations of Suśruta Samhita in the eighth century were not from Sanskrit but from a Persian version of Suśruta.†

It is most interesting to note that according to Magnus[10] the Persians knew cataract under the name *mautika bindu* which, according to Oppert, means "a drop of pearl" in Sanskrit. Today, as I learned from an Indian colleague, in some regions cataract is popularly known as *motya*, meaning "pearl."

In summary, the Hindu surgeons regularly employed two instruments: At first a short, sharp lancet for perforating the sclera, which was withdrawn as soon as the resistance of the latter was overcome, and then a thin cylindric needle with a blunt end, some 12.5 mm. long and three-edged in shape (the edges being blunt), with which gentle pressure was exerted on the superior edge of the lens until the zonule was broken and the cataract dislocated. The Arabian surgeons also frequently used two instruments, while the Greeks had one and the same sharp needle for both steps. Discussed later will be why the procedure with two instruments must have been the more cautious one and a better guarantee for a favorable result; injuring the capsule (and the lens) with the sharp instrument favored iridocyclitis and secondary glaucoma which were the worst complications of the procedure, perhaps more so than hemorrhage, retinal detachment and the subacute and somewhat rarer fulminating infection.

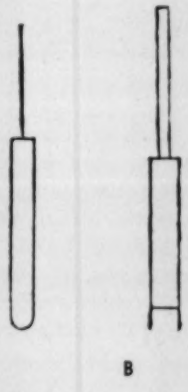


Fig. 7 (Feigenbaum). (A) Round needle (*mihatt mudawwar*). (B) Hollow needle (*mihatt mugawwaf*). Khalifa.

* Indian works on medicine have been translated early into Arabic, partly through the intermedium of Persian, and they are quoted in the *Continens* (Hirschberg[8]).

† In Arabic it appeared as Ketab as-Susrud. Another treatise, that of Vagbhata I (Aśtangahridaya Samhita) had also been translated into Arabic at the same period as the *Surgery* of Sindhi (of Bagbhat).

These grave sequelae and unfavorable results of the "contemporary" operation by quacks are carefully exposed by Elliot in his book on the *Indian Operation of Couching*[3] in which the southern variety, the so-called Mohammedan operation (performed by the *vaidyan*), is chiefly dealt with. Of greatest interest to us from his large experience is: (a) his description of the operating procedure used by the contemporary Indian coucher (this quack is known in the various parts of India as the *suttya* or *mal*, as the *rawal*, or as the *vaidyan*); (b) his statistics with regard to the results of the treatment (the collection of the 780 cases under review took 12 years); and (c) his pathologic findings in the 54 globes excised. To quote:

(a) The operators appear to rely largely on manual dexterity, and to aim at completing the procedure in a minimum of time. (Whether it is the anterior operation [couching performed through the cornea with a needle or a sharp thorn] or the posterior operation [fig. 10] which has been carefully studied and described by one of Elliot's assistants, Dr. Ekambaram. The latter's original description of the method will well repay a careful perusal. He speaks of the operators as being ambidextrous and very skilful. Their surgical equipment for the operation consists of a small lancet-shaped knife, guarded to within a few millimeters of its tip by a roll of cotton-wool, wrapped round it for the purpose, and of a copper probe four inches long and about 1.5 mm. in diameter (fig. 11). A cotton thread twisted round this probe at a spot 12 mm. from its point serves the same purpose as the stop in the Bowman's needle. From the point to this stop the instrument is triangular in section. The patient is directed to look well toward the nose, and the surgeon then gently marks out the selected spot by pressing with his thumbnail on the conjunctiva covering the sclera, about eight mm. out from the cornea, and about two mm. below the horizontal meridian. In some cases the operator steadies the eye by firm digital pressure exerted through the partly everted lower lid. He next takes his lancet in his hand, and it will be observed from the illustration that it might easily pass for a roll of cotton-wool; this, indeed, is what the patient is led to believe it really is. To heighten such an impression, the point is covered with a sandalwood paste, prepared beforehand coram publico, with a good deal of ostentation. The patient is informed that this "cataract-cleansing drug is about to be applied to the eye," and under cover of the suggestion the operator plunges the lancet through the tunic of the globe at the spot already selected. The alarm thus occasioned is allayed by the assurance that the "medical application" is over. The copper probe is next produced, and is inserted through the wound up to its stop, being held between the thumb and two fingers; a circular movement is given to its point, the stop resting against the puncture and serving as a pivot for the movement. According to Ekambaram, the object is to tear through the suspensory ligament from behind. Immediately following this step, a downward stroke of the point is made in order to depress the now loosened lens."

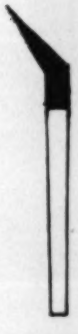


Fig. 8 (Feigenbaum). Lancet (Khalifa) for puncturing the outer coats.

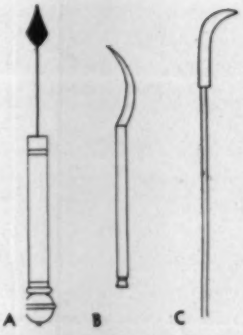


Fig. 9 (Feigenbaum). (A) Lancet-shaped *al-barid* for puncturing the outer coats (Abulqasim). (B) Sickle-shaped *al-barid* (Abulqasim). (C) Al-barid (anonymus, *Escorial Codex*).

As to the coucher and his habits "all who have seen them at work (fig. 12) agree that their methods are dirty and septic to a degree, and the oft-expressed wonder has ever been, not that their results are so bad, but that they are ever good."

(b) Lieut. Colonel E. F. Drake-Brockman . . . estimated the coucher's successes at not more than 10 percent. Lieut.-Colonel Henry Smith went so far as to declare that even the best cases, if followed long enough, ended in absolute blindness. Lieut. Colonel F. P. Maynard formed a much less unfavorable estimate of the coucher's results (46 percent of successes). . . . As to Elliot's own statistics: "In only 10.59 percent was the vision (always corrected with lenses) 1/3 and upward. In another 11.05 percent the vision was 1/2 to 1/10;

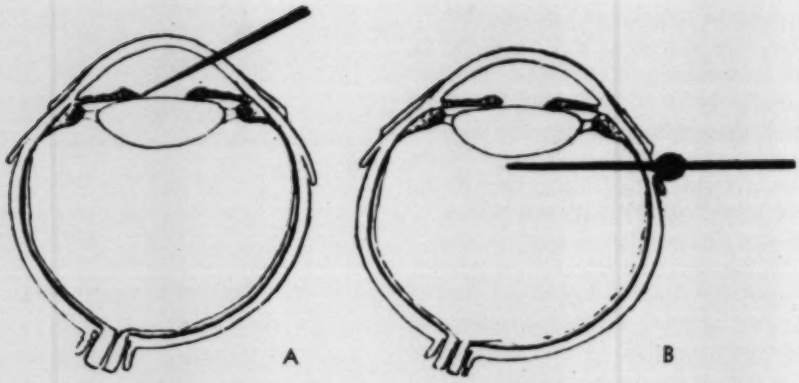


Fig. 10 (Feigenbaum). Indian couching operation. Anterior (A) and posterior (B) approach (Elliot).

in 9.64 percent it was 1/10 to 1/50, and in 7.05 percent it was finger counting at two feet or less. If every case that got a vision of 1/10 and upward be considered a success, the coucher can claim 21.64 percent. Again if anything from 1/10 vision to the ability to count fingers close to the face be counted as partial success, the figure for this class is 16.69 percent. . . . The table shows the causes of failure. . . . The figure for iritis and iridocyclitis comes to 35.76 percent of the *total* number of cases; glaucoma accounts for 11.05 percent, imperfect dislocation of the lens for 8.04 percent, retinal detachment for 3.53 percent, optic atrophy for 2.59 percent. . . . It is clear that the native coucher undertakes a certain number of what we should recognize as inoperable cases.

(c) . . . It is obvious that (in the pathologic series) we are dealing with the couchers' failures alone, and are excluding his successes. In a very large percentage of the latter the lens is seen, during life, to be floating in the vitreous, apparently untrammelled by adhesion . . . and . . . without any sign that the intraocular tension is raised. (The study of the 54 globes dealt with) has shown that the fixation [to the intraocular tissues] or otherwise of the lens is a question of the amount of septic action set up by the operation. . . .

The various dislocations of the lens, some of them almost incredible, found to have taken place after couching, as well as the frequent accidental injuries to structures other than the lens, are discussed.* Inflammation of the uvea, mostly plastic and confined to the iris and ciliary body, very greatly varied in intensity (sympathetic ophthalmia being extremely rare):

"In a number of specimens the evidence of inflammatory action was either absent or only to be detected on very careful examination."

Hemorrhages into the vitreous were rarely seen in vivo, because the patients mostly presented themselves at a late stage. While usually insisting on "the trail of septic infection of the eyeball by penetrating wounds" caused by the coucher, Elliot

* Here one is vividly reminded of the warnings expressed by Hunain ibn Ishaq.

Fig. 11 (Feigenbaum). Lancet and copper probe of Indian coucher (Elliot).

admits that sometimes inflammation may have "been induced by chemotaxic substances."

Retinal detachment (mostly extensive and complete) was found in 38 of the 54 globes, in the very great majority caused undoubtedly due to traction from within, by shrinking exudative membranes. Nineteen out of the 54 globes showed evidence of high intraocular pressure, which figure obviously cannot "be taken as a reliable index of the numerical frequency of glaucoma as a complication of the operation. . . ." In 17 of the 19 the angle of the anterior chamber was extensively closed, and in 3 of these the chamber was almost reduced to a potential slit.

It was necessary to quote rather extensively from Elliot's monograph since it contains a severe condemnation of the mischievous practice of couching for cataract, because of its importance as a rare and reliable analysis and its value as a historic document. Considering, however, the question before us, the material presented and the conclusions drawn therefrom must be seen in their proper light. When discussing the surgical method of needling, practiced at a time before a safer method was devised for dealing with blindness from cataract, in antiquity and even in later periods, it is not possible to make a straight-forward comparison of the procedure as practiced by the ancient specialists (at least some of them) and by the Indian quack of today. There is a considerable difference between them even apart from the fact that, in times of old, there was no alternative, and the coucher, especially the one belonging to the better class, filled during the ages some function in society.

Though the pathologic conditions described by Elliot seem strongly to suggest an infective origin, this is certainly not so in every case. Mechanical and chemical irritation of a tissue and antigenic reaction to lens protein are frequently enough the decisive noxious factors. The less the lens is injured the less damaging probably is the effect of the irritating lenticular substance within the globe.

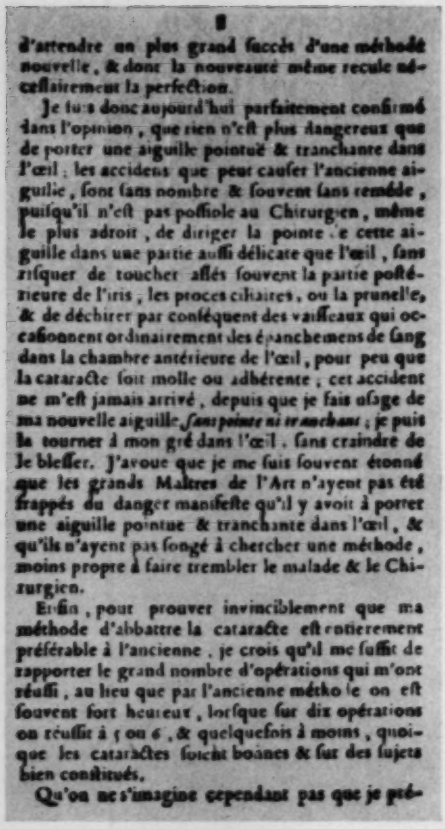
Without discussing the matter any further, I shall cite only some passages from Duke-Elder's *Textbook* which contains the experience of a great many observers on the reaction of the eye to dislocation of the lens as a whole or of lenticular masses within the vitreous.

Fig. 12 (Feigenbaum). Indian coucher at work (Elliot).

At first it (the posteriously dislocated lens) is usually movable but eventually organized membranes tend to anchor it. Provided the capsule has not been ruptured, the eye may tolerate the lens in this position without apparent ill-effects for some years. . . . In posterior dislocations . . . the end result is usually a gradual loss of vision owing to degenerative and glaucomatous processes which, however, may require a considerable number of years for their fruition.

In search for further confirmation of the assumption that there must be a marked difference as to the final result, between the procedure with two instruments and the "simplified" one, with the perforating needle alone, I came upon an inestimable testimony which could almost be classed as experimental. Before finally building up his method of extraction, Daviel, already on the way to formulating the steps necessary for this purpose, still hesitating or not yet fully determined, tried to improve the method of depression. In a letter to Dr. de Joyeuse of Marseille, Daviel[31] writes:

I shall report to you on several eye operations performed since 7th November, 1746, the day I arrived in Paris, truthfully, no matter whether they



d'attendre un plus grand ſuccès d'une méthode nouvelle, & dont la nouveauté même recule néceſſairement la perfection.

Je ſuis donc aujourd'hui parfaitement confirmé dans l'opinion, que rien n'eſt plus dangereux que de porter une aiguille pointuë & tranchante dans l'œil; les accidens que peut cauſer l'ancienne aiguille, ſont ſans nombre & ſouvent ſans remède, puiſqu'il n'eſt pas poſſible au Chirurgien, même le plus adroit, de diriger la pointe de cette aiguille dans une partie auſſi délicate que l'œil, ſans riſquer de toucher aſſés ſouvent la partie poſtérieure de l'iris, les proces ciliaires, ou la prunelle, & de déchirer par conſéquent des vaiſſeaux qui occaſionnent ordinairement des épanchemens de ſang dans la chambre antérieure de l'œil, pour peu que la cataracte ſoit molle ou adhérente; cet accident ne m'eſt jamais arrivé, depuis que je fais uſage de ma nouvelle aiguille *ſans pointe ni tranchant*; je puis la tourner à mon gré dans l'œil, ſans craindre de le bleſſer. J'avoue que je me ſuis ſouvent étonné que les grands Maîtres de l'Art n'ayent pas été frappés du danger manifeſte qu'il y avoit à porter une aiguille pointuë & tranchante dans l'œil, & qu'ils n'ayent pas ſongé à chercher une méthode, moins propre à faire trembler le malade & le Chirurgien.

Enfin, pour prouver invinciblement que ma méthode d'abbattre la cataracte eſt entierement préférable à l'ancienne, je crois qu'il me ſuffit de rapporter le grand nombre d'opérations qui m'ont réuſſi, au lieu que par l'ancienne méthode on eſt ſouvent fort heureux, lorſque ſur dix opérations on réuſſit à 5 ou 6, & quelquefois à moins, quoique les cataractes ſoient bonnes & ſur des ſujets bien conſtitués.

Qu'on ne s'imagine cependant pas que je pré-

Fig. 13 (Feigenbaum). Page from Daviel's letter, dealing with the advantages of using two instruments in cataract depression.

have been successful or whether the results were not according to my wishes.

Speaking of the erroneous views current among laymen and even among surgeons about the alleged safety of the needling operation, Daviel observes:

As far as I am concerned I think differently. It is only after much experimentation on eyes at autopsy and a great number of operations performed on living people, having always in mind to improve the method of depression of cataract, that I learned all about the doubtful points at issue which may arise. . . . The operation is by far not as safe and as easy as many believe and as I thought myself when I began to practice it. . . . No matter how good the cataract may be, how perfect the health of the patient and how able the surgeon operating, symptoms frequently appear which the most experienced oculists would have the greatest difficulty to explain; sometimes the cataract surges again after the best performed operation, at another time lacrimation sets in; now vomiting shortly afterwards occurs, now swelling of the conjunctiva and the eye, agonizing pain in the whole eyeball with severe headaches, and finally suppuration of the whole organ, which no care of the most able surgeon can avert or check. These are observations I have made following my operations and following those I have seen performed by famous oculists. . . . These difficulties . . . made me look out since a long time for ways of preventing them, means gentle, easy & safe at the same time.

In the following a case is described in detail where a second difficult intervention on the same eye was made with a special needle.

I contrived a needle which I still use today, without point or cutting edge (the operation mentioned resulted in loss of the eye because of suppuration). . . . But this failure did not deter me from pursuing my idea and from continuing experimentation on the eyes of cadavers in order to bring my new method to a point of safety which I think it has reached today . . . and I dare-say my new way of operating was so fortunate that I depressed 7 cataracts one after the other with every success possible and without any mishap. . . . The cure of the 7th patient, whom you know . . . has won me great fame . . . since my arrival in Paris I was consulted by some 400 patients, on over 200 of whom I performed operations for cataract as well as for other ocular affections. . . . In 75 cataract operations, most of them . . . (presenting all kinds of difficulties) . . . I had the good fortune to succeed in 61; I do not think one could expect* a better success from a new method . . . I am thus perfectly convinced today that there is nothing more dangerous than introducing into the eye a pointed and cutting needle. The bad complications which can be produced by the old needle, are innumerable, and they mostly cannot be redressed, since it is impossible even for the most dexterous surgeon to direct the point of that needle . . . without risking frequently enough to hit the posterior surface of the iris, the ciliary processes or the pupil and thus to tear vessels which causes bleeding. . . . Such an accident has never again occurred to me, since (after having perforated the tunics of the eye with a small lancet) I make use of my new needle which has no point and no cutting edge and which† I can turn within the eye ad libitum, without having to be afraid of an injury. To tell the truth, I am often surprised that the great masters of the Art have not noticed the obvious danger of introducing into the eye a pointed and cutting needle. . . .

* Here starts the page depicted in Figure 13.

† Daviel's improved instruments for depressing cataract (taken from *Mercure de France*, April, 1750, p. 26) are reproduced in Hubbel's article.[36]

Finally to prove convincingly that my method of cataract depression is preferable to the old one, I think it will suffice to refer to the great number of successful operations (reported above), while with the old one, one often had to be quite satisfied, if out of 10 operations 5 or 6 were successful or even less (in otherwise most favourable cases). You should not imagine, however, that I maintain my method to be infallible . . . or free from every danger; it has its faults. . . .

. . . I performed an extraction of a cataract still situated in the posterior chamber of the right eye of M. Garion. . . . The observations made in this operation have evoked in me great ideas about extraction of the cataract. . . .

Having done already all I could in order to depress the cataract of this patient and being unable to succeed in any other way, I determined to open the lower part of the clear cornea; in order to introduce my needle easier into the posterior chamber of the eye, I for quite a while held the cornea everted by means of a small forceps, and let the lens get out through the opening just made; though the aqueous humour ran out altogether, and so did a small quantity of the vitreous, this did not prevent the patient from seeing all the objects shown to him and to recognize people.

. . . Sir, will you please note the significance of this eye operation, as the point in question is a cataract extracted from the posterior chamber of the eye and not from the anterior; there are several observations reported on the latter by the late M. de St. Yves . . . but this famous author did not mention extraction of cataract from the posterior chamber. . . .

A description follows of a long series of operations performed. Appended to the letter are testimonials from personages of high rank to the good results obtained by Daviel by his treatments (how strange!).

In one respect Daviel was mistaken. His method, as we know, was not new—it seems to have been forgotten and was revived by him. Even in the 17th century there were now and again authors (for example the Dutchmen Smalzius; Nuck, 1692) who performed the depression with two needles, one pointed and slightly canaliculated, the other blunt altogether (Magnus). Albinus (in 1695) depicted similar ones in his treatise *de cataracta*.[8]

Having examined the material necessary to a reasoned judgment, my conclusions can now be summarized. It is easy to imagine how and why the Indian method may have been taken over inaccurately by the Greeks. Having no experience with the procedure (and not knowing, of course, what implications an injured lens had for the final result of the operation) but being sometimes able to get along with one instrument only, it can be imagined that they thought this deviation made no difference. Was not the main object of the surgeon to free the pupil from the obstructing matter? It can even be imagined that enthusiastic novices acquired the idea that the use of two instruments was a mere whim of the strange Easterners, and that confining oneself to one instrument was a useful simplification. The fact that the Alexandrians must have thought the operation to be an easy one is mirrored in the optimistic expressions of Celsus.

Thus, the essential difference in technique between the Indian procedure and the "simplified" method that was brought over to the Mediterranean shores in the fourth or third century B.C. is (a) apt to explain to a considerable degree the unfavorable results, the growing dissatisfaction with the method of the more conscientious Greek physicians, their unwillingness to use it, and the ensuing stagnation. On the other hand it is (b) in my opinion a sure indication and a further evidence of the evolvement of the Indian procedure very early in antiquity which originally—possibly in prehistoric times—must have, of course, started with one instrument. But, how many centuries must it have taken the Indian surgeon to find out that the introduction of a second instrument, a blunt one, was the safer way of operating?

These arguments cannot properly be concluded without paying homage to Julius Hirschberg (1843-1925), my late teacher. To him, our main authority in the history of ophthalmology, the letter of Daviel, herein quoted, was well known and so were most of the facts with regard to the use of one instrument by the Greeks, of two by the Hindus, and that the latter method was taken over by some of the prominent Arabs. The question arises why did not he himself arrive at the conclusions which seem so cogent to

me? The reason for this, or so it appears, is not difficult to find; it is an historical one: Hirschberg flourished at a period when the conceptions of anaphylaxis, allergy, and immunity were either not yet born or (at the beginning of our century) in their early infancy. He therefore could not be particularly concerned with the chemotactic reactions of the intraocular tissues to the lens protein of the depressed cataract. The dangers of couching for cataract in his view must have been about the same as conceived by Elliot, namely injuries, infection, and their direct sequelae.

4. Epilogue: Developments up to the modern cataract operation

After the decline of Arab medical art, from the 13th century on, a period of stagnation set in. Because of its dangers the method of couching gradually was abandoned by the medical profession. In the hands of quacks in Europe who, along with other "simplifications," used only one instrument—if instrument it could be called—the results obtained could only be worse than those obtained by the Indian coucher in our day. Here and there honest attempts were made by solid craftsmen, as, for example, the itinerant surgeons Bartisch (16th century) in Germany and Banister (17th century) in England, but charlatans prevailed.

In his book, *Augendienst*,[32] Bartisch describes in a trenchant way the habits of the itinerant quack-couchers:

> There are in our midst today very many who pretend to be oculists and eye-doctors. They get their patients and perform on them the needling operation in the market-place, in the wind outdoors, in front of everybody . . . and allow them to leave like a hog from the trough. . . . These people usually become blind.

And emphasizing their negligence with regard to the instrument they use, he says: ". . . they sometimes take just a small piece of wood or a little stick into which they fix a common sewing needle." Bartisch himself used a needle made of silver (fig. 14-b) which he recommends always to keep clean.

Significantly, in 1612 the *Statutes of Avignon* permit everybody to practice couching, stone cutting and operation for hernia without examination, because they simply need nothing else but practice. Only at the beginning of the 18th century, after recognition of Michel Brisseau's statement that couching for cataract was depressing the opaque lens, a new period began with the physicians becoming encouraged to perform the operation without being afraid of injuring the lens (Heister, 1713, fig. 14-c). A new class came up of "learned" couchers of whom Taylor and Woolhouse became famous and—notorious. We arrive at a period when the idea of extracting the lens from within the eye could mature in Daviel's mind.

After this innovation of extracting the cataract from the eye (in 1748) instead of the ancient risky method of couching, which with time, obviously due to negligent and ig-

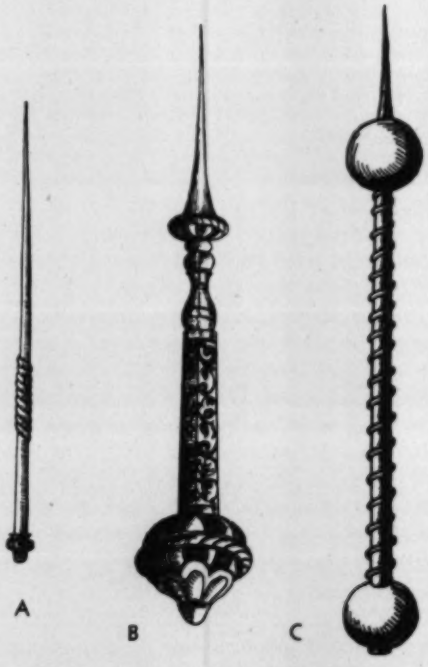


Fig. 14 (Feigenbaum). Couching needles of: (A) Celsus (according to Andrea a Croce, 1560); (B) Bartisch (1583); and (C) Heister (1713). The latter two instruments have Renaissance and Barock ornamented handles (Greeff[38]).

norant execution, has become of ill repute (even before something better could be offered in its stead), it is surprising to find that it took another century before couching was definitely given up as a rival method of extraction, and this in spite of Daviel's successes, then quite unusual, in some 89 percent of his cases.

At the end of the 18th century and in the first quarter of the 19th century (and even somewhat later) some surgeons of repute actually tried to reinstall the depression operation to its former place, suggesting all kinds of improvements, or simply praising the simplicity of its performance. The reason for this obstruction was mainly conservatism on the part of a large section of the profession although it is true that in the pre-anesthetic period, a good deal of dexterity was required of the surgeon in handling the eye and the patient; and there was still the undeniable danger of infection. (We have learned from the letter of Daviel how he himself must have hestiated before becoming firmly convinced.)

It is noteworthy, however, that the lengthy discussions of the pros and cons had at least one positive result: procedures were elaborated of how best to deal with soft cataracts by discission.*

Finally, mention must be made of some "precursors" of Daviel, as it were, in the occasional extraction of cataract through paracentesis of the cornea. In Galen's work on the *Method of Therapy* a famous phrase reads: "Some have tried to empty these (the hypochymata), as I shall discuss in the treatise of surgery."† There seems to be no doubt about the meaning of this sentence, as it comes directly after the author had said that hypochyma differed from other pathologic structures in the body (that have to be destroyed) in that it has only to be shifted (by needling) from one place (the pupillary area) where it is an obstacle to vision, to another less important one. Nothing is said here about the conditions under which this was done, whether these were (as it would appear from some descriptions) cases of hypopyon or fragments of a cataract which, in the course of a couching operation entered into the anterior chamber. Paracentesis of the cornea had in such instances (luxation of the whole lens or lenticular fragments into the anterior chamber) actually been done in the beginning of the 18th century before Daviel, by St. Yves and Petit.

There is also one famous Greek surgeon in antiquity, who is said to have extracted cataract, Antyllus (second or third century); his writings on surgery are lost, but his description of cataract operation, as already mentioned, is actually preserved in an Arabic translation. Rhazes (9th-10th century) quotes him in his *Continens:* "Antilis. And some people split the lower part of the pupil and extracted the cataract; and he says: this is possible when the cataract is thin (better translated into "soft"), and when it is thick (better: "hard") it cannot be done; because the albuminous humour would gush out with the cataract."*

But it was left to Daviel to establish extraction as a sound, planned procedure. His method marked a new era and has been improved upon since, step by step. In the first half of this century, the technique was greatly advanced by being practiced more and more as an intracapsular extraction, a perfection inaugurated mainly by Colonel Smith in 1905,† remarkably enough, in India, the country which for a number of factors was singled out to become the birthplace of the cure for cataract in antiquity as well as a fertile soil for the more recent perfection of the modern operation for this condition.

15, Abyssinian Street.

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* E. C. Conradi was one of the first (1797) who inaugurated a rational and simple method for needling soft cataracts, namely splitting of the anterior lens capsule alone.[8]

† This treatise is not preserved or may, possibly, not have been written.

* Translated by Hirschberg and coworkers[8] from the Arabic (*Escorial Codex*).

† In 1910 Henry Smith had 24,000 extractions to his credit of which 20,000 were intracapsular (Garrison[88]).

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THE PATHOGENESIS AND PATHOLOGY OF OCULAR ONCHOCERCIASIS*

PART IV. THE PATHOLOGY

F. C. RODGER, M.D., M.CHIR.
Oxford, England

In the introductory part of this section of the thesis, a number of points are briefly summarized, as they have been more fully covered in preliminary publication.† In particular may be mentioned the original papers describing the microscopic appearance of the parasite during the stages of its dissolution within the eye (Rodger[1]), and the description of the routes preferred by the embryos of O. volvulus in entering the various structures of the eye (Rodger[2]). Throughout this part, which describes the pathology of the ocular manifestations of onchocerciasis, one must be constantly aware of the presence of the parasites, or of fragments of them, either alive at the time the eye was excised (as the staining will show) or, being dead, in different stages of disorganization, for it is that background, as much as that of the structural patterns formed, which makes the topic unique. By showing the mutations of the parasite body after it dies, and the places where it is most likely to be found in the eye, I hope to create this necessary background before the detailed pathology is considered.

INTRODUCTION

1. MATERIAL

The observations were carried out on 12 eyeballs and 37 pieces of tissue; for various reasons another eight eyeballs were rejected in the final assessment, for it was essential to use material that was clinically above criticism and without any complicating feature whatever. The 12 best and most characteristic eyes only were selected. They included acute, subacute and chronic stages of the various clinical lesions of onchocerciasis. The same strictures were applied to the biopsy material.

2. TECHNIQUES

Some of our specimens were sent to the Department of Pathology in the Institute of Ophthalmology, London, where celloidin embedding was performed, and routine sections (hematoxylin-eosin) supplied. Others (the majority) were sent to the Department of Histology in the University Laboratory of Physiology, Oxford, where low-viscosity nitrocellulose embedding was carried out. On my return from Africa I was able to use the facilities in Oxford, and a great variety of staining techniques was undertaken; the

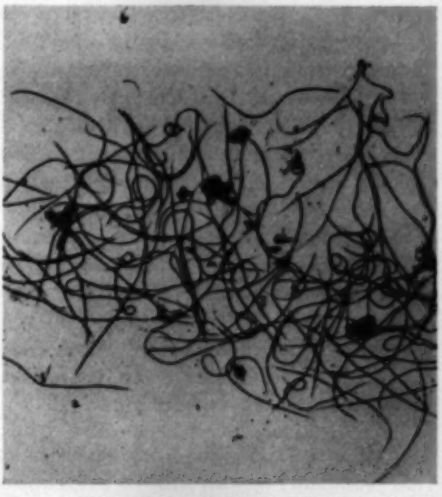

Fig. 12 (Rodger). Microfilariae of O. volvulus in skin biopsy one by two mm. (Mayer's hemalum. ×55.)

* From the Visual Research Unit, Oxford University.

† The editorial committee of the *Transactions* of the Royal Society of Tropical Medicine and Hygiene is warmly thanked for permission to reproduce the photomicrographs from the earlier publications referred to in this paragraph.

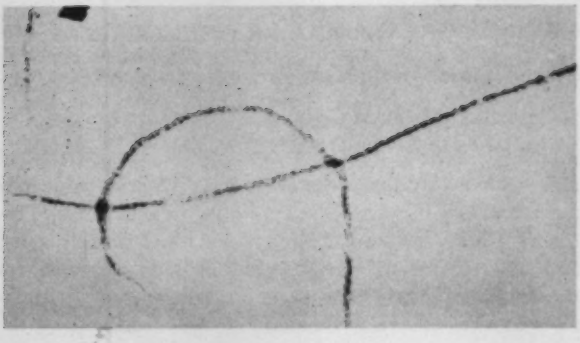

Fig. 13 (Rodger). Microfilariae of O. volvulus. (Mayer's hemalum. ×220.)

London blocks were permitted to be brought to Oxford and further sections cut there at leisure. Those who have carried out research will understand how appreciative any worker is when a colleague generously gives him of his time and service. I owe a great debt of gratitude to Dr. Ashton of London and Mr. Leach of Oxford in this connection. The latter elaborated a method of bleaching the pigment in the heavily pigmented negroid eye without affecting the staining, and this proved of great value, making the location of the microfilariae easier (Chesterman and Leach[3]). Considering that the eyeballs after they were excised in the bush had to be rushed to the nearest airport—perhaps 400 miles away over difficult terrain—they were in remarkably good condition when they reached the United Kingdom; only occasionally were there signs of hardening. The specimens were fixed in Bouin for two days and dispatched in 70-percent alcohol. Biopsies were sent the same way but were paraffin-embedded.

The photomicrography was carried out partly in Oxford by E. H. Leach and partly in India by me.

Routine stains used included phloxine tartrazine, Mallory, Masson, van Gieson, Weigert's and periodic acid-Schiff.

3. The dissolution of microfilariae of O. volvulus in the human eye and its effect on the tissues

Here we shall describe the microscopic changes which occur in microfilaria O. volvulus (apparently after its death) in the tissues of the eye. We shall also attempt to associate these changes with the local reactions appearing in the vicinity of the parasite.

The cornea is the tissue best suited to observations of this kind, especially in pigmented eyes. Because the parasites and inflammatory cells are required to travel through a comparatively simple avascular structure, the changes can be viewed as in a culture medium. Recently and lightly infected eyes are usually more informative than old heavily infected ones as the latter

Fig. 14 (Rodger). Microfilaria streptocerca. (Mayer's hemalum. ×220.)

present a complicated picture in which acute and chronic tissue changes overlap, and both living and dead microfilariae in every stage of dissolution exist side by side.

The observations reported here are presented with some diffidence for fragments of a microfilaria are difficult to photograph, the whole seldom being in focus. The absence of color further weakens the pictorial evidence so necessary in work of this kind. However, although these are insoluble defects, they are after all only relative ones.

To date it has been presumed that it is the products of disintegration of O. volvulus which produce inflammatory changes in the human eye. Robles[14] and Calderon[15] suggested that a toxin secreted by the adults or the larvae when alive was responsible for the pathologic picture. Later Shafi[6] and Vogel[17] inclined to the same view; the former, who is often quoted, made no study of onchocerciasis, simply drawing a parallel with helminthic parasites.

Strong[18] suggested the reaction was at least partly due "to the passage of large numbers of microfilariae through the lymphatic vessels of the eye and the subsequent dilatation and oedema which results." Later[19] he stated "at the same time it would appear probable that the microfilariae through their metabolism, or their death and destruction in the tissues, might also exert a pathologic effect."

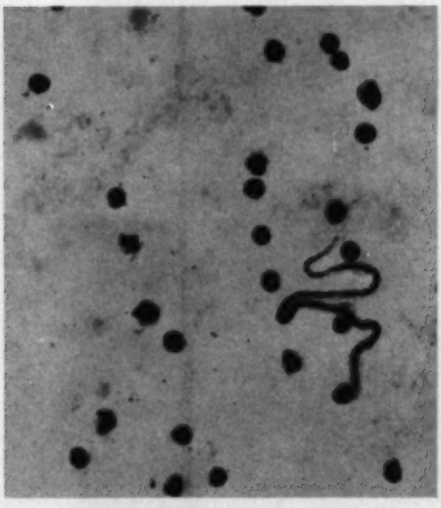

Fig. 15 (Rodger). Microfilaria perstans. (Giemsa, R66. ×220.)

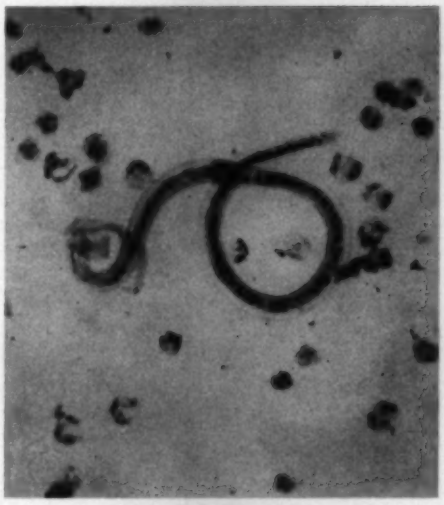

Fig. 16 (Rodger). Microfilaria bancrofti. (Ehrlich's hematoxylin. ×220.)

The finding of microfilariae in every ocular tissue logically proves nothing except that the adults are probably not implicated. We cannot even be sure of this.

Hissette[10] summed the position up carefully thus: (1) there is a slight and slow reaction to the living microfilariae, and (2) a clear and appreciable reaction to the dead larvae. The former, as has been suggested by Strong, might be due simply to the passage of the parasites in number; equally well it might be due to a toxin which they or the adults secreted; the latter, as Hissette believed, is most likely to be caused by the disintegrative products of the larvae. These conclusions, nevertheless, were all equivocal.

What is surprising is that no pathologist to date has been able to associate the reactions of the tissues with microscopic changes in the microfilarial bodies. Hissette came very near to this for he was aware that some microfilariae stain well, every nucleus being

Fig. 17 (Rodger). (A) Microfilaria loa, Ehrlich's hematoxylin, ×220. (B) Bleached iris revealing fragment of microfilaria below the posterior epithelium, not likely to be visible without bleaching, phloxine tartrazine, ×510. (C) Unbleached negroid iris, showing how impossible it is in the darker Negro races, at any rate, to find microfilariae in this structure, ×510.

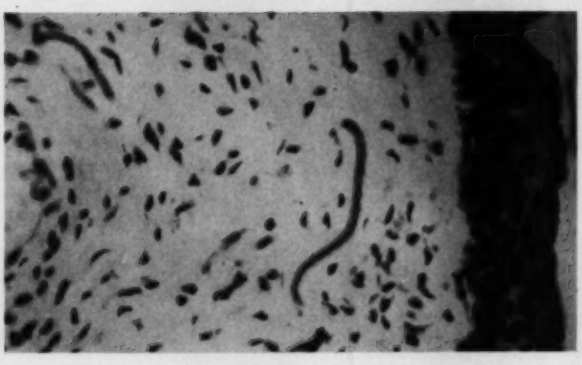

Fig. 18 (Rodger). Microfilariae of O. volvulus in lid. (Hematoxylin-eosin, ×300.)

clearly outlined, whereas others appear to lose their affinity to the stain, the nuclei becoming masked. He assumed the former were alive at the time the eye was excised, and the latter dead. Unfortunately he did not attempt or was unable to associate these two different appearances of the parasite with the processes of inflammation.

Where the body outline is uniformly cylindric, the nuclei well-stained and clear-cut, the rest of the soma unstained (hematoxylin-eosin) or only faintly stained (phlox. tartr.), the microfilaria can be said to have been alive at the moment the eye was excised; in a recently invaded eye (that is, with no inflammatory reaction visible) this is the typical appearance.

The changed appearances of the microfilaria as it is absorbed, presumably after death, have classified as under:

Stage 1. Increased affinity of the soma to the stain occurs. This may be called "somatic staining."

Stage 2. The nuclei then become indistinct, the difference in color intensity between them and the soma becoming less and less until the whole stains uniformly. The body characteristically is irregularly swollen. This stage has been called "ballooning."

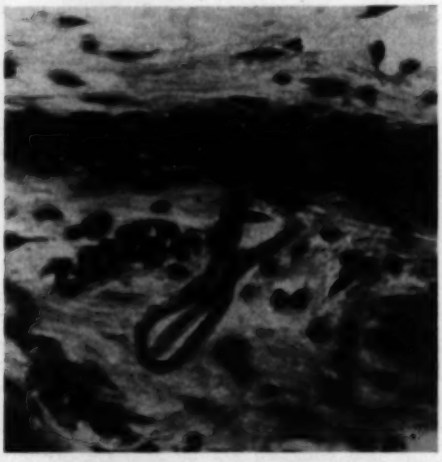

Fig. 19 (Rodger). Microfilaria of O. volvulus at limbus. (Hematoxylin-eosin, ×280.)

Fig. 20 (Rodger). Microfilaria of O. volvulus in corneal pannus. (Masson, ×220.)

Stage 3. The body now breaks up abruptly into fragments. This stage has been called "fragmentation."

Stage 4. Finally, in proportion as the fragments become smaller and fewer, the surrounding tissue stains diffusely blue (hematoxylin-eosin) or pinkish-blue (phlox. tartr.) I have called this the stage of "diffusion and absorption" as that is what appears to be happening.

These changes are almost certainly those of autolysis for at no time were they found associated with the presence of macrophages or giant cells.

No reaction was seen until "ballooning" occurred (Stage 2). A few eosinophil leukocytes were invariably closely applied to the parasite body at this stage; with fragmentation (Stage 3) their number increases enormously, and a few round cells can be seen. The local cyto-architecture probably determines the number and dispersal of the inflammatory cells. In the potentially wide space between the epithelium and Bowman's membrane they cluster round the parasite in large numbers; in the more dense substantia propria the inflammatory cells, required to pass along the narrow interstitial spaces, are not so closely applied to the disintegrating body. At the start of Stage 4 the picture is variable; sometimes eosinophil leu-

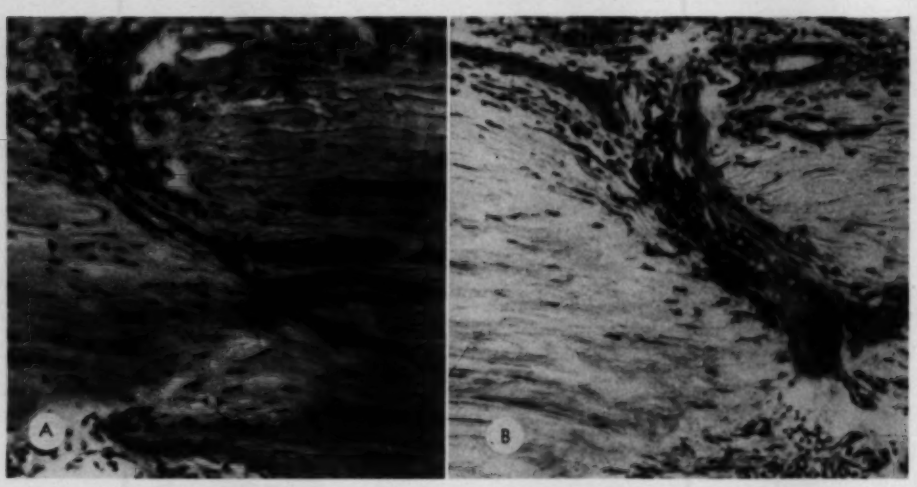

Fig. 21 (Rodger). (A) Microfilaria of O. volvulus passing down in sheath of perforating anterior ciliary vessel. (B) Taken through the lumen of the same vessel. (Masson, ×440.)

kocytes dominate (they are always present), sometimes fibroblasts; the plasma cells and lymphocytes have also increased in number. Coinciding with the arrival of these migratory cells into the cornea, new capillaries grow toward the parasite body: during Stage 4 the vessels are often already close to the inflammatory focus. As the limbal area has usually been the site of earlier more advanced inflammation, there is a reservoir of the different cells within easy access.

By the time the microfilarial fragments have become completely absorbed the number of plasma cells invariably equals or exceeds the eosinophil. On the whole, however, it is the fibroblasts of the stroma which are most conspicuous, supporting the view that we are dealing with a reactive fibrosis induced by the stimulus of non-necrotising toxins derived from the parasite body as a result of of its dissolution.

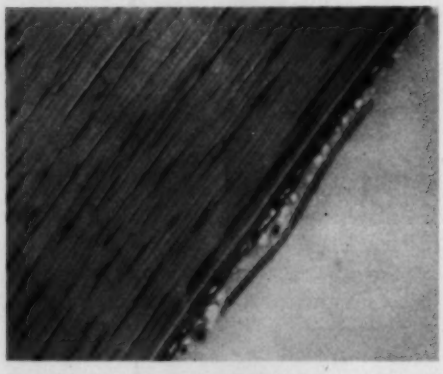

Fig. 22 (Rodger). Microfilaria of O. volvulus in anterior chamber. (Phloxine tartrazine, ×220.)

It is not possible to say how long the process of dissolution takes. The inferences are that it lasts only for a few days. The acute reaction may continue for some days longer; although the nature of the inflammatory cells is more in keeping with a chronic lesion even where the clinical signs were acute, that is a commonly found anomaly. There seems little doubt that after the parasite body has disintegrated the toxic products linger on diffusing very slowly from the primary focus into the neighboring tissues. In this way adjacent parts may be affected without necessarily being the locus of a dead parasite; such areas it is believed may be distinguished by the frequent absence of eosinophil leukocytes; a single capillary associated with a few fibroblasts and plasma cells is a common finding at some distance from a parasite in Stage 3 or 4 of dissolution. It is impossible of course to prove that no

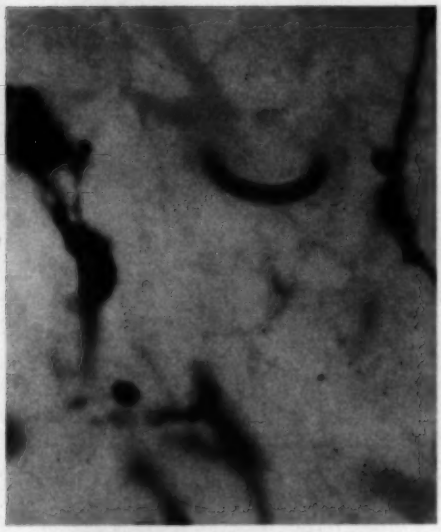

Fig. 23 (Rodger). Microfilaria of O. volvulus in iris (Phloxine tartrazine, ×500.)

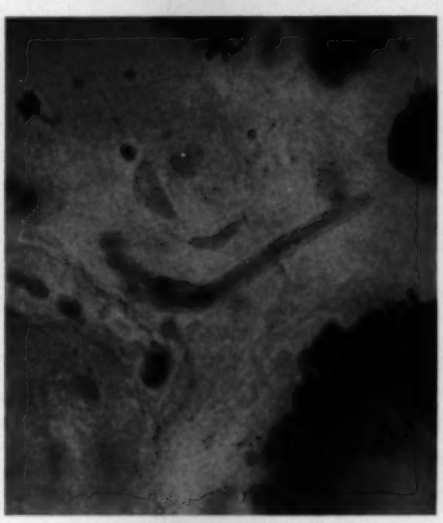

Fig. 25 (Rodger). Microfilaria of O. volvulus in ciliary body. (Phloxine tartrazine, ×500.)

parasite died in such small areas of pannus; that many arise as a result of the diffusion of toxic products from a nearby focus is a personal view.

There was no strong evidence of a transitory stage between ballooning and fragmentation; that is why fragmentation is described as occurring "abruptly". That an intermediate stage occurs is likely. Possibly Stage 4 might come in for some criticism; the faint staining of the tissues which characterizes it has not been seen in any other condition except, perhaps, calcification in an old keratitis. The association in an infected eye of a local aggregation of eosinophil leukocytes, fibroblasts, and plasma cells with diffuse staining of the tissues in which these cells lie is highly suggestive, nevertheless.

One concludes that O. volvulus is a good example of a perfectly adapted parasite, and

Fig. 24 (Rodger). Microfilaria of O. volvulus in vitreous. (Masson, ×440.)

Fig. 26 (Rodger). Microfilaria of O. volvulus in choroid. (Masson, ×350.)

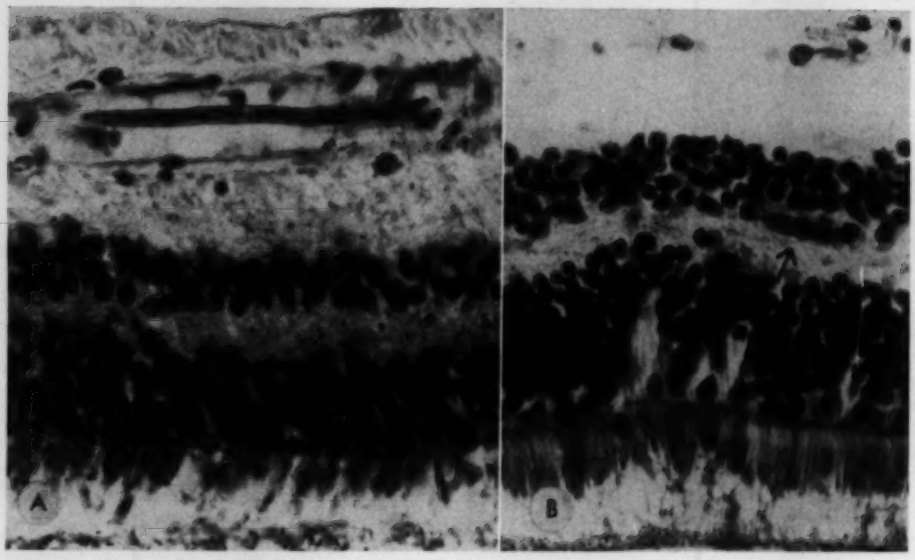

Fig. 27 (Rodger). (A) Microfilaria of O. volvulus in retina. (B) From a neighboring section in the same eye. (Phloxine tartrazine, ×550.)

that the inflammatory changes observed in the cornea in this study were caused by the products of dissolution of mf. bodies, and not by any exotoxin secreted by them or the adult worms when alive.

4. THE MOVEMENTS OF THE PARASITES INTO AND WITHIN THE EYE

Lids to outer coats of eyeball. The microfilariae in a case with a high I.D.F.* (Rodger and Brown[11]) can always be found in great numbers in the lids. Palpebral and bulbar conjunctival biopsies in such cases reveal the same picture. Indeed, as has been suggested elsewhere (Rodger[12]) skin biopsies of the lid are as revealing as conjunctival biopsies. In these patients the anterior segment of the eye is invariably heavily invaded (cornea, corneoscleral junction, and root of iris) as are the extraocular muscles, the loose episcleral tissues around Tenon's capsule and the outer fibers of the sclera. The latter is the usual site of microfilariae as far as the sclera is concerned; most photomicrographs reporting microfilariae in the sclera in fact reveal them either in the episclera or just inside the outer coat; only rarely have we found microfilariae in the substance of the sclera itself, except anteriorly at the limbus where the structure is looser and scleral fibers intermingle with corneal. In view of the resistance of the sclera proper it is not surprising the microfilariae do not penetrate it readily.

Outer coats of eyeball to inner. There is a quantitative decrease usually in the number of parasites found in the uvea in heavily infected eyes as we move back from the limbus to the optic nerve. This is such a common finding it suggests that in such eyes the scleral barrier is a very real one except anteriorly. In other words, it appears that the microfilariae penetrate wherever the tissues are least resistant; that is to say the routes most likely to be taken are from under bulbar conjunctiva into the cornea between its epithelium and Bowman's membrane, in a posterior direction through the corneoscleral

* I.D.F. = Individual Density Figure.

junctional fibers to the angle of the eye and hence into the uvea, or into the corneal stroma, in that order. That the most readily accessible route is above Bowman's membrane is borne out by the observations we have made on nearly 3,000 heavily infected eyes with the ocular biomicroscope, where subepithelial corneal microfilariae were seen many times but seldom if ever stromal; moreover it is the classic site of pannus onchocercosus. The paragraph following enlarges on the possible anterior routes.

Inner coats of eyeball to intraocular fluids. It was not possible to find any evidence in the sections suggesting the mode of passage of microfilariae into the aqueous humor. It seems likely, nevertheless, that when they reach the angle of the eye they will break through the thin uveal scaffolding forming the ligamentum pectinatum comparatively easily, and that when they are present in the iris they will emerge through its fine anterior endothelium with even less difficulty. We do not believe microfilariae can pass from the retina directly into the vitreous, as when they were present no sign of this was seen; however, it is possible they might. More probably, in my view, the parasites break through the anterior face of the vitreous gel, which affords no barrier at all, having previously passed from the anterior chamber into the posterior.

Fig. 29 (Rodger). Microfilaria in cornea, somatic staining. (Phloxine tartrazine, ×500.)

Passage via the adventitial sheaths of blood vessels. The presence in some eyes of a restricted anterior invasion associated with a restricted but definite choroidal invasion suggested that some parasites must pass into the choroid by another route as a quantita-

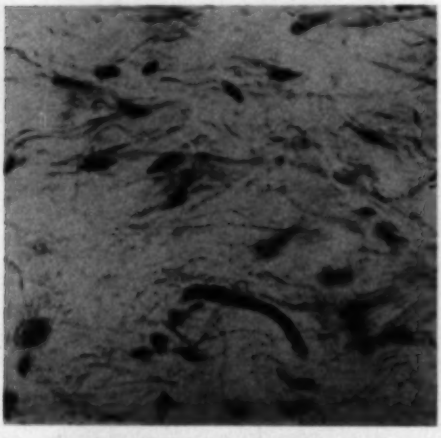

Fig. 28 (Rodger). Microfilaria of O. volvulus in optic nerve. (Phloxine tartrazine. ×440.)

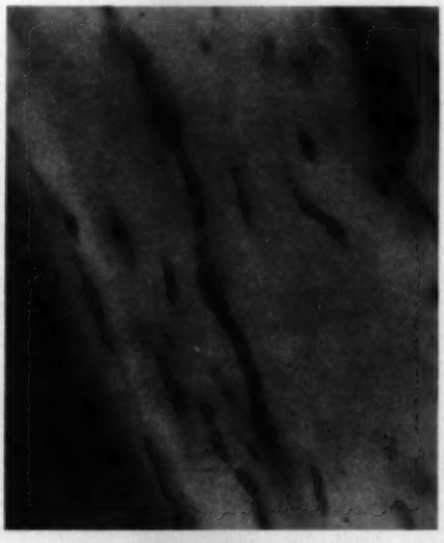

Fig. 30 (Rodger). Ballooning microfilaria in cornea. (Hematoxylin-eosin, ×500.)

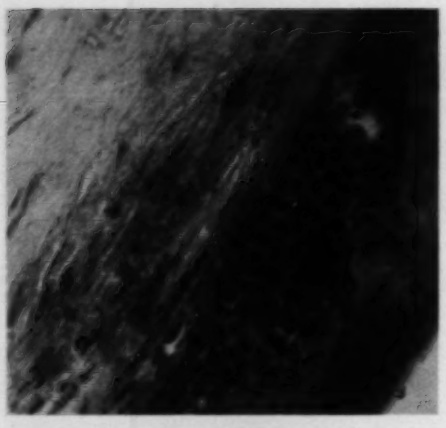

Fig. 31 (Rodger). Fragmented microfilaria in cornea. (Hematoxylin-eosin, ×500)

tive decrease from cornea to optic nerve is to be expected in an invasion from the anterior segment backward. Because it was fairly certain that the hard wall of the sclera afforded an almost impassable barrier, the possibility was considered that the microfilariae might pass down the loose fibrous sheaths of the penetrating equatorial vessels. A certain amount of evidence of this was found in serial sections. At the same time it was abundantly demonstrated that the adventitial sheaths of the perforating anterior ciliary vessels were even more favored by the parasites as an easy means of passing from the outer eye to the inner, thereby adding to the routes of entry into the eye at or around the limbus. Theoretically, although it is possible for microfilariae to reach the choroid without invasion of the anterior segment, as it will depend on orbital invasion which is unlikely to happen except where the parasites are also present in force at the limbus, choroidal invasion probably occurs only rarely as an isolated phenomenon. Our clinical experiences support this belief.

Invasion of the retina by the microfilariae of O. volvulus. In the course of the last-mentioned observations the original discovery was made that microfilariae invade the retina. The parasites apparently are able to reach this structure by forcing a passage through the pigment epithelium for there was evidence of such a break-through. There was no sign, however, of a break in the continuity of the membrane of Bruch. So apparently they must either break through from the ciliary body between the pigment epithelium and the cuticular lamina of Bruch or pass from the vitreous directly into the retina. One of two microfilariae observed lay in the sheath of a retinal blood vessel with its head projecting into the lumen in the direction of the optic disc. The attitude and position suggest it had not passed up into the retina via the vascular sheath of a retinal vessel (unlikely although not impossible) but had entered the sheath from within the retina, and was in the process of forcing its way along the loose adventitial coat. The position of its head apparently within the lumen of the vessel may be an artefact. The distinct staining suggests this parasite was alive at the time of the operation.

Photomicrographs give a rough indication only of the size of such microfilariae by comparison with the retinal nuclei. The only other unsheathed species which volvulus might be confused with is microfilarial perstans, although the latter is smaller. Factors

Fig. 32 (Rodger). Microfilaria in cornea, dissolution and absorption. (Hematoxylin-eosin, ×220.)

other than size, which anyway is an unsatisfactory standard where only a fragment of the parasite is visible, depend upon the part of the body that can be made out. In the more complete of the two microfilariae seen in the retina the head and half the body are visible, so that by the shape and distribution of nuclei identification can be made with confidence, apart altogether from the evidence of its size. In neither of the two retinal microfilariae was the tail visible, but the evidence supplied by the caudal nuclei is not important where the other characteristics described above exist. More convincing still, perhaps, is the fact that both patients had an I.D.F. of 31 and that in none of 10 thick blood films examined in each were blood-borne microfilariae found. In short all the evidence led to the one conclusion, that these microfilariae in the human retina were volvulus.

Invasion of the optic nerve. Although finding in two other subjects a solitary microfilaria in the optic nerve substance there was no evidence to show (as is likely) that they burrowed into the nerve from the orbit along the septal nutrient vessel sheaths or (less probably), through the border tissue of Elschnig from the choroid or sclera. In each of the two positive cases there was massive orbital invasion, so invasion via the blood vessels of the nerve is quite a probability.

With this brief account of what the observer is likely to see with the microscope in sections of an eye invaded by the embryos of O. volvulus, we shall now pass on to a detailed pathology of the various ocular lesions.

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BLEPHAROPTOSIS, CONGENITAL AND ACQUIRED*

Relationships of Anatomy and Pathologic Physiology to Surgical Correction

Ralph S. Hamilton, M.D.
Memphis, Tennessee

Definition and Derivation

The word blepharoptosis is derived from the Greek—blepharo, meaning lids, and ptosis, to fall. Blepharoptosis may be defined as the inability to elevate the upper lid, the causation of which is some pathologic situation which diminishes or abolishes the effective action of the levator palpebrae superioris muscle.

Accompanying Pathologic Conditions

Blepharoptosis is a most serious, and often complicated situation, which for many reasons demands correction; and in most instances surgical correction. The cosmetic

* From the Wills Eye Hospital and the Graduate Hospital of the University of Pennsylvania, Philadelphia. This thesis was submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania toward the requirements for the Degree of Master of Medical Science (M.Sc. (Med.)) for graduate work in ophthalmology.

blemish alone certainly is adequate reason for correcting this pathologic situation. Probably in most instances, this is the most compelling reason for correction. However, there are many other conditions which frequently accompany blepharoptosis and which will in themselves necessitate correction of the blepharoptosis and the accompanying pathologic conditions. Some of these accompanying pathologic conditions are:

1. Curvature of the spine from elevating the chin in an attempt to see below bilateral blepharoptosis.
2. Amblyopia of an eye constantly covered by a drooping lid.
3. Amblyopia due to suppression from an accompanying vertical or horizontal strabismus.
4. Lack of opportunity for development of single binocular vision.
5. Paresis or paralysis of one or both superior rectus muscles—a frequent accompliment of blepharoptosis and a reasonable occurrence when one remembers the close embryologic, anatomic, and innervational association of the superior rectus muscle and the levator palpebrae superioris muscle.
6. Other types of horizontal or vertical strabismus.
7. Unsightly facial grimace resulting from continued contraction of the occipitofrontalis muscle attempting to elevate the blepharoptotic lid.
8. Epicanthus or blepharophimosis.

Certainly any one of these situations may gravely affect a person's mental and physical adjustments to life.

This study was undertaken in an attempt to correlate the anatomy and pathologic physiology to the most rational form of surgical correction; and to report the results obtained from these various techniques in a series of 74 blepharoptotic lids.

The methods of surgical correction of blepharoptosis will be dealt with more extensively later. Suffice it now to cite the necessities for correction and to say that these require surgical correction in a majority of instances. Perhaps the only cases having no surgical component are hysterical blepharoptosis, blepharoptosis associated with active, advancing central nervous system or orbital pathology, and blepharoptosis associated with metabolic dysfunction.

Anatomy of Levator Palpebrae Superioris

Wolff has very clearly described the anatomy, that is, the origin and course, of the levator palpebrae superioris as "arising from the under surface of the lesser wing of the sphenoid above and in front of the optic foramen by a short tendon which blends with the underlying origin of the superior rectus muscle." Wolff also states that "the muscle is a flat ribbonlike muscle belly which passes forward below the roof of the orbit and on the superior rectus muscle to approximately one centimeter behind the septum orbitale where it ends in a membranous expansion or aponeurosis. This aponeurosis then spreads out to give the whole muscle the form of an isosceles triangle."

Spaeth describes the aponeurosis of the levator palpebrae superioris insertion as follows: "the levator is attached to the orbicularis palpebrarum by an upper lamella which overlies the anterior surface of the tarsus to form a potential space. This upper lamella also sends some fibers to the orbital septum and to the non-striated muscle fibers. A middle lamella of the tendon aponeurosis is attached to the posterior surface of the tarsus, and some reflected fibers to the underlying superior rectus muscle. The more clearly defined lowermost lamella is inserted into the superior conjunctival cul-de-sac."

There is a lateral horn of the levator palpebrae superioris muscle which passes through the lacrimal gland defining the orbital and palpebral portions of this gland. This lateral horn then inserts into the orbital tubercle and upper portion of the lateral palpebral ligament. The medial horn attaches below the frontolacrimal suture and to the medial palpebral ligament.

From posterior to anterior or conjunctival to skin surfaces, the layers of tissues encountered in cross section of the upper lid just at the superior margin of the tarsus are: conjunctiva, Mueller's muscle, levator (vertical fibers), tendon, orbicularis muscle (horizontal fiber bundles) and skin.

The innervation of the levator palpebrae superioris muscle is from the superior division of the third cranial nerve, as is the innervation of the superior rectus muscle. The innervation of these two muscles composes the superior division of the third cranial nerve. The innervation to the occipito-frontalis muscle (with which one is concerned in surgical correction of blepharoptosis) is the seventh cranial nerve.

Etiology of blepharoptosis

The etiology of blepharoptosis may be divided into two categories: (A) congenital blepharoptosis, and (B) acquired blepharoptosis. In the series to be presented later, 50 cases or 74 eyes were studied preoperatively, operatively, and postoperatively. There were 28 cases of congenital blepharoptosis, of which 15 cases were bilateral, or 30 eyes; and 13 cases were unilateral, or 13 eyes; making a total of 43 eyes with congenital blepharoptosis. In this series of 74 eyes there were 22 cases of acquired blepharoptosis, of which nine cases were bilateral, or 18 eyes; and 13 cases were unilateral or 13 eyes; making a total of 31 eyes with acquired blepharoptosis.

My conclusions as to the etiology of congenital blepharoptosis from the preoperative, operative, and postoperative study of these 43 eyes with congenital blepharoptosis are that congenital blepharoptosis may be due to central ectodermal nuclear aplasia, other central ectodermal defects, peripheral mesenchymal aplasia of the levator palpebrae superioris muscle, misdirection of fifth cranial nerve fibers into the levator palpebrae superioris, or to fibrosis of the levator palpebrae superioris with or without fibrosis of the intraocular muscle.

Associated superior rectus muscle weakness may give one a clue to central ectodermal nuclear or other defects. This may be further confirmed by surgical exploration which reveals normal levator palpebrae superioris and superior rectus muscles. Surgical exploration of the levator palpebrae superioris muscle is the diagnostic feature of peripheral mesenchymal defects of aplasia of fibrosis. Examination of the patient, asking him to utilize the muscles innervated by the fifth cranial nerve (usually the muscles of mastication) while carefully watching the blepharoptotic lid may lead one to the diagnosis of misdirection of fifth cranial nerve fibers into the levator palpebrae superioris muscle.

Spaeth has given us the best and most complete etiologic classification of acquired blepharoptosis. This classification is quoted below with Dr. Spaeth's permission.

Class I. Traumatic peripheral

A. Peripheral—complete or incomplete section of the levator.

B. Peripheral cicatricial with the levator uninvolved, but scar formation preventing normal amplitude of movement. This may be skin, cul-de-sac or both.

C. Peripheral cicatricial, from conjunctival and muscle involvement as with symblepharon from chemical or hot metal burns.

D. Peripheral from destruction of bone tissues as in osteomyelitis, or loss of soft tissues of upper lid, or both.

E. Blepharoptosis appearing after reconstruction of a socket.

Class II

Traumatic central and cerebrospinal third cranial nerve paresis or paralysis or a cervical sympathetic paralysis caused by section of the nerve.

Class III

Peripheral, inflammatory or neoplastic, the result of such intraorbital pathology as foreign body in orbit, pseudotumor, neurofibroma or mucocele.

CLASS IV. ATONIC BLEPHAROPTOSIS

A. Blepharoptosis of senility

B. Blepharoptosis of blepharochalasis

C. Atonic blepharoptosis after old simple enucleation where there is no Tenon's capsule implant. Here one finds a dry atrophic socket with flabby, relaxed upper lid.

CLASS V. NEUROMUSCULAR DISTURBANCES

A. Myasthenia gravis

B. Myasthenia of thymic tumor

C. Ophthalmoplegias of thyrotoxicosis. Here one is more likely to see lid retraction than blepharoptosis. All three of these entities frequently show paresis or paralysis of the superior rectus muscles.

CLASS VI

Blepharoptosis of cervical sympathetic involvement or Horner's syndrome.

CLASS VII. BLEPHAROPTOSIS OF III CRANIAL NERVE PARALYSIS

A. Peripheral extracranial (wounds, etc.)

B. Infranuclear, but intracranial (cranial injury leading to the pseudo-Graefe syndrome). Also may be seen with perichiasmal pathology, or an aneurysm near or of the circle of Willis.

C. III cranial nerve blepharoptosis from nuclear and intranuclear conditions, as inflammations, vascular lesions, degenerations, muscular dystrophy, syphilis, and so forth.

CLASS VIII

Blepharoptosis associated with pseudo-Graefe syndrome.

CLASS IX. HYSTERICAL BLEPHAROPTOSIS

A. Paralytic

B. Pseudoptosis of blepharospasm

Thus in summary one can see that acquired blepharoptosis may be: (1) peripheral in the levator palpebrae superioris, (2) extracranial but intraorbital, (3) central intracranial, (4) sympathetic, (5) metabolic, (6) atonic, (7) cicatricial, (8) degenerative, (9) inflammatory, or (10) hysterical.

The various causes of acquired blepharoptosis are tumors, traumas, infections, vascular diseases and metabolic dysfunctions. One can see that the causes, and the location of these causes make acquired blepharoptosis somewhat complex and often a diagnostic challenge. It is imperative that the etiologic cause for acquired blepharoptosis be known and either cured or permanently stabilized before any surgical correction be considered.

SURGICAL CORRECTION OF BLEPHAROPTOSIS

From the foregoing parts of this thesis it is evident that blepharoptosis may be a complex, complicated condition caused by a multitude of etiologic factors, whether the blepharoptosis be congenital or acquired. A large majority of these factors must be corrected surgically. As was previously stated the only cases of blepharoptosis having no surgical component are hysterical blepharoptosis; blepharoptosis associated with active advancing central nervous system or orbital pathology; and blepharoptosis associated with metabolic dysfunction.

The ideal results for which one should strive in the surgical correction of blepharoptosis are: (1) to attain a lid which opens voluntarily while awake and closes involuntarily to protect the cornea during sleep; (2) to have a normal blinking reflex; (3) to have a lid fold corresponding to that of the opposite lid; (4) to have equal vertical and horizontal palpebral fissures; and (5) to have no hypotropia or diplopia. In a sentence one must always strive for a good cosmetic and functional result.

The first step in attempting to achieve these results is a careful preoperative study and history of each case of blepharoptosis in order to determine the etiology. A good history and family history are important in determining whether blepharoptosis is congenital or acquired. Congenital blepharoptosis and blepharoptosis associated with blepharophimosis not infrequently are found in several generations of a family.

Objective examination should include a visual acuity in each case, amblyopia is a frequent accompliment of complete unilateral blepharoptosis. The vertical width of each palpebral fissure should be measured, as should the horizontal length of each palpebral fissure. This horizontal length may be especially helpful in blepharophimosis.

Care should be taken to measure the width of the palpebral fissure with and without occipito-frontalis muscle elevation of the lid. To accomplish this, the patient may be instructed not to wrinkle the brow. If the patient is unable to co-operate due to a conditioned reflex of attempting to elevate the lids in this manner, firm pressure with the examiner's fingers over each eyebrow will prevent lid elevation from occipito-frontalis contraction.

A complete muscle balance examination should be done with special attention being directed to the action of the superior rectus muscles. Congenital blepharoptosis associated with superior rectus muscle paresis or paralysis is not an infrequent finding, having been reported in from 29 percent to 47 percent of some series of cases.

A cycloplegic refraction should be done in all cases, and certainly in those cases of blepharoptosis complicated by horizontal strabismus. A careful evaluation of the presence or absence of levator function is most imperative. Presence or absence of a lid fold should be noted.

In Table 1 are presented the 50 cases and 74 eyes which were studied in the preparation of this thesis. The cases may be grouped as follows: bilateral, 24 cases or 48

TABLE 1

SURVEY OF CASES STUDIED

Case	Age (yr.) Race & Sex	Diagnosis	Surgical Procedure	Results
1. H.B.	29 W.F.	Left blepharoptosis with Horner's syndrome following cervical sympathectomy for Raynaud's disease	Blepharoptosis is very mild, no surgery performed	—
2. D.M.	5 W.F.	Marcus-Gunn jaw-winging reflex, O.S.	Levator tenotomy, Reese orbicularis transplant, Berke's superior rectus muscle resection and attachment to tarsus	Complete blepharoptosis, undercorrection of blepharoptosis, inadequate cosmetic and functional results. Child with 35D.right hypertropia prior to surgery, now has 40 D. right hypertropia
3. C.B.	2 W.M.	Possible Marcus-Gunn, O.D.	No surgery as yet, blepharoptosis very mild, child young and diagnosis not certain as yet	—
4. G.D.	3 W.M.	Left congenital blepharoptosis with some levator function	Blaskovics levator reaction and tarsectomy, further levator resection	Under correction Good cosmetic and functional result
5. M.McG.	3 W.M.	Bilateral congenital blepharoptosis with levator function, O.U.	Bilateral Blaskovics levator resection and tarsectomy	Good cosmetic and functional result, O.U.
6. J.H.	3 W.M.	Bilateral blepharoptosis with bilateral blepharophimosis	Bilateral Blaskovics levator resection and tarsectomy and epicanthal surgery	Good cosmetic and functional results, O.U.
7. B.B.	67 W.M.	Bilateral atonic senile blepharoptosis	No surgery	—

TABLE 1 (*Continued*)

Case	Age (yr.) Race & Sex	Diagnosis	Surgical Procedure	Results
8 R.B.	27 W.M.	Bilateral congenital blepharoptosis with horizontal and vertical strabismus, some levator function, O.U., atrophic levators at surgery	Blaskovics levator resection and tarsectomy, O.D.	Undercorrection
			Further levator resection, O.D.	Good cosmetic and functional results, lid slightly lower than, O.S.
			Blaskovics levator resection and tarsectomy, O.S.	Good cosmetic and functional result
			Recession left inferior oblique	
			Resection right medial rectus	
			Resection left inferior rectus	
9. B S.	3 W.M.	Complete congenital III cranial nerve paralysis external and internal, O.D.	Hunt-Tansley procedure recession of lateral rectus, tuck inferior oblique, O.D., resection medial rectus, resection advancement superior rectus, O.D.	Fair cosmetic and functional result, good result first two months then lid came down approximately 1.5 mm.
10 J M.	15 W.F.	Blepharoptosis, O.S. after removal dermoid of orbit	Blaskovics levator resection and tarsectomy	Good cosmetic and functional result
11 S.S.	4 W.F.	Bilateral congenital blepharoptosis with blepharophimosis	Bilateral Blaskovics levator resection and tarsectomy with epicanthal surgery	Good cosmetic and functional result, O.U.
12. N.I.	58 W.M.	Left blepharoptosis with some slight levator function and paralysis of left medial rectus muscle following probable cerebral vascular accident	Blaskovics levator resection, O.S.	Good cosmetic and functional result of lid. Patient complained of some diplopia for two months then apparently learned to suppress, O.D.
13. F.C.	28 W.F.	Bilateral congenital blepharoptosis with no detectable levator function, levator anatomically good muscle tissue at surgery	Bilateral levator resection with tarsectomies	Excellent cosmetic result, patient has some exposure of corneae, slight epithelial punctate staining with fluorescein, this cleared objectively and subjectively with use of 1% methyl-cellulose at bedtime for one month
14. A.B.S.	62 W.M.	Blepharoptosis following transfrontal craniotomy for suspected sphenoidal ridge meningioma, no levator function, O.S.	Everbush levator resection, O.S.	Inadequate cosmetic and functional result
15. D.K.	19 W.M.	Bilateral congenital blepharoptosis with some bilateral levator function	Bilateral Blaskovics levator resections and tarsectomies	Good cosmetic and functional result, O.S., slight undercorrection, O.D., fair cosmetic and functional result, O.D.

TABLE 1 (*Continued*)

Case	Age (yr.) Race & Sex	Diagnosis	Surgical Procedure	Results
16. E.P.	80 W.F.	Complete blepharoptosis following Kreenlein orbitotomy for pseudotumor of the orbit, left, no levator function present	Hess sutures, O.D.	Fair cosmetic and functional result
17. K.B.	55 W.F.	Senile blepharoptosis with old contracted socket following enucleation, O.D.	Tarsectomy with plastic wedges under periorbita of floor of orbit	Good cosmetic result from tarsectomy
18. W.E.	8 W.F.	Blepharoptosis of reconstructed lid in case of congenital anophthalmos, O.D.	Hess sutures	Fair cosmetic result
19. B.P.	7 W.F.	Bilateral blepharophimosis with intermittent exotropia	Bilateral Blaskovics levator resections and tarsectomies with bilateral epicanthal surgery	Good cosmetic and functional results, O.S.
20. L.N.	61 W.M.	Complete blepharoptosis, O.U., with no levator function following encephalitis, postencephalitic neuritis	Hess sutures, O.U.	Good cosmetic and functional results, O.D. Good cosmetic and fair functional result, O.S. Some corneal exposure, O.S.
21. M.G.	44 W.F.	Bilateral progressive muscular dystrophy of all extraocular muscles	Bilateral Blaskovics levator resections and tarsectomies	Good cosmetic and functional results, O.U., six months later
22. T.G.	33 W.M.	Neurofibromatosis, O.S., with complete blepharoptosis and no levator function present	Hess sutures	Fair cosmetic and functional result
23. F.C.	70 W.F.	Senile atonic blepharoptosis, O.U. Partial, O.D., complete, O.S.	Wheeler blepharoptosis surgery (orbicularis fascia sutured to superior rectus muscle)	Good cosmetic and functional result, patient has some hypotropia and diplopia in upward gaze
24. S.P.	6 W.F.	Congenital blepharoptosis, O.S., with some levator function	Blaskovics levator resection and tarsectomy	Good cosmetic and functional result
25. R.F.	5 W.M.	Bilateral congenital blepharoptosis, much more, O.S. Levator function, O.D.; slight, O.S.	Blaskovics levator resection and tarsectomy, O.S.	Good cosmetic and functional result
26. R.P.	63 W.F.	Progressive muscular dystrophy of all extraocular muscles	No surgery	—
27. K.V.	15 W.F.	Congenital blepharoptosis, O.D., with slight levator function	Hess sutures, and repeated nine months later	Inadequate correction fair cosmetic and functional results
28. B.P.	4 W.F.	Congenital blepharoptosis, O.D., with levator function	Blaskovics levator resection and tarsectomy	Inadequate cosmetic result
29. J.G.	8 W.M.	Treacher-Collins syndrome with blepharoptosis, O.S.	Blaskovics levator resection and tarsectomy, more levator resection one year later combined with Kuhnt Szymanowski for entropion of left lower lid	Inadequate cosmetic result Inadequate cosmetic result

TABLE 1 (*Continued*)

Case	Age (yr.) Race & Sex	Diagnosis	Surgical Procedure	Results
30. A.D.	74 W.F.	Bilateral blepharoptosis from progressive muscular dystrophy of levator and all intraocular muscles	Friedenwald-Guyton suture for left upper lid	Good cosmetic and functional result
31. D.H.	5 C.M.	Congenital blepharoptosis of left upper lid without levator function	Reese orbicularis transplant	Inadequate cosmetic result
32. R.F.	16 W.M.	Blepharoptosis left upper lid, complete paralysis of levator from accident, possibly levator and superior rectus severed or superior division of III cranial nerve cut, no levator function	Hess blepharoptosis sutures	Inadequate cosmetic result. Hess sutures to be repeated
33. J.H.	16 W.F.	Bilateral congenital blepharoptosis with blepharophimosis	Bilateral Blaskovics levator resection and tarsectomy	Good cosmetic and functional result
34. C.S.	70 W.F.	Bilateral blepharoptosis associated with Mikulicz's syndrome from lymphosarcoma of lacrimal and parotid glands	No surgery	Patient's blepharoptosis disappeared on ACTH and nitrogen mustard as lacrimal gland receded to normal size
35. A.O'N.	48 W.F.	Complete III cranial nerve paralysis, O.D.	Craniotomy revealed no central nervous system lesion	Following craniotomy patient developed improvement in III cranial nerve paralysis
36. R.L.P.	37 C.F.	Old enucleation with blepharoptosis and some levator function, O.D.	No surgery	—
37. J.S.	47 C.M.	Complete internal and external ophthalmoplegia on left with left IV cranial nerve and left VI cranial nerve paralyses	No surgery	—
38. A.E.	6 W.M.	Blepharoptosis on left with some levator function	Stevenson's modification of Everbush operation	Good cosmetic and functional result
39. K.A.	20 mos. C.M.	Complete blepharoptosis, O.D., with no levator function	Hess sutures	Fair and cosmetic and functional result
40. E.S.	48 W.M.	Bilateral progressive muscular dystrophy of extraocular muscles and levators	Hess sutures, O.U.	Inadequate permanent cosmetic result, some exposure, good cosmetic result
41. R.O'D.	4 W.F.	Bilateral congenital blepharoptosis with slight levator function	Bilateral levator resection through skin	Good cosmetic and function result
42. W.S.	3 W.M.	Complete congenital blepharoptosis, O.S., with no levator function	Hess sutures	Good cosmetic and functional result
43. D.O.	3 W.M.	Congenital complete blepharoptosis with no levator function, O.U., no superior rectus function O.U.	Hess sutures, O.U.	Fair cosmetic and functional result

TABLE 1 (*Continued*)

Case	Age (yr.) Race & Sex	Diagnosis	Surgical Procedure	Results
44. D.B.	3 W.F.	Bilateral congenital blepharoptosis with levator function exophoria	Bilateral Blaskovics levator resection and tarsectomy	Good cosmetic and functional result, O.U.
45. F.C.	42 C.M.	Blepharoptosis of left upper lid following old enucleation	Reese orbicularis transplant	Inadequate cosmetic and functional results
46. R.K.	2 W.M.	Bilateral congenital blepharoptosis with good levator function	Bilateral Hess sutures	Fair cosmetic and functional results
47. R.O.	2 W.M.	Bilateral congenital blepharoptosis with blepharaphimosis	Epicanthal surgery, bilateral Friedenwald-Guyton sutures	Good cosmetic result. Fair cosmetic and functional result, O.S., inadequate result, O.D.
48. E.H.	73 W.F.	Bilateral senile atonic blepharoptosis following bilateral cataract surgery, keratitis sicca	Crutch glasses	—
49. C.N.	24 W.F.	Unilateral congenital blepharoptosis of left upper lid with levator function and normal superior rectus muscle function	Blaskovics	Exposure keratitis
50. P.O'D.	3 W.F.	Bilateral congenital blepharoptosis with levator function, O.U.	Blaskovics	Good cosmetic and functional results

eyes; unilateral, 26 cases or 26 eyes; congenital, 28 cases or 43 eyes (bilateral, 15 cases or 30 eyes; unilateral, 13 cases or 13 eyes); acquired, 22 cases (bilateral, 9 cases or 18 eyes; unilateral, 13 cases or 13 eyes).

The cases of congenital blepharoptosis may by divided into: (1) bilateral or unilateral blepharoptosis with some levator function; (2) bilateral or unilateral with no levator function; (3) congenital blepharoptosis associated with blepharophimosis; and (4) congenital blepharoptosis associated with the Marcus-Gunn jaw winking reflex.

The cases of acquired blepharoptosis all fall into the complete classification of acquired blepharoptosis previously quoted from Spaeth.

The results of the above operative cases were graded as follows: A *good* cosmetic and functional result was given to the case if the lid was elevated to completely expose the pupillary aperture, to close covering the cornea during sleep, and to have a lid fold simulating that of the opposite eye. A *fair* cosmetic and functional result was given to the case if there was some improvement in elevation of the lid, but not enough to expose completely the pupillary aperture; or if there was some overcorrection resulting in some exposure of the cornea during sleep or while awake. An *inadequate* cosmetic and functional result comprised those cases without improvement or those cases which were overcorrected and resulted in serious corneal exposure.

In the surgical correction of blepharoptosis, one has three muscles to utilize: (1) the occipito-frontalis muscle, (2) the superior rectus muscle, and (3) the levator palpebrae superioris muscle.

Table 2 summarizes the surgical results in these cases.

There were 23 occipito-frontalis muscle

TABLE 2

SUMMARY OF SURGICAL RESULTS

Type Procedure	Number of Eyes	Good	Fair	Inadequate
Occipito-frontalis	22 (23 procedures)	4	13	6
Superior rectus	2 (2 procedures)		1	1
Levator	35 (38 procedures)	30	2	6
TOTALS	59 (63 procedures)			
No surgery	15			

surgical procedures performed. Of these 23 procedures four or 17.4 percent were good cosmetic and functional results, 13 or 56.5 percent were fair cosmetic and functional results, and six or 26.1 percent were inadequate cosmetic and functional results.

The number of superior rectus muscle surgical procedures is inadequate from which to draw conclusions. The advantages and disadvantages of superior rectus muscle surgery will be presented from the work of other men in the field of blepharoptosis.

There were 38 levator muscle surgical procedures performed. Of these 38 procedures 30 or 79 percent were graded as good cosmetic and functional results, two or 5.2 percent were fair cosmetic and functional results and six or 15.7 percent were inadequate results.

From these situations in this series of 63 operative procedures for the surgical correction of blepharoptosis one is able to conclude that the highest percentage of good cosmetic and functional results was obtained with levator resection and partial tarsectomy. Also these procedures yielded the fewest number of fair cosmetic and functional results and the fewest number of inadequate cosmetic and functional results.

From the series of procedures utilizing the occipito-frontalis muscle, one is able to conclude that the lowest percentage of good cosmetic and functional results was obtained; that the highest percentage of fair cosmetic and functional results was obtained; and that the highest number of inadequate cosmetic and functional results was obtained.

Every case of blepharoptosis does not lend itself to judicious correction with levator muscle surgery. Thus superior rectus muscle surgery or occipito-frontalis muscle surgery must be employed.

The results of this series would imply that one should do levator muscle surgery as the procedure of choice, if the indications for this are present. These indications will be brought out later. One may also conclude that levator muscle surgery may be repeated with satisfactory results if necessary. One may also deduct that if it is necessary to utilize the occipito-frontalis muscle, one should not expect as high a percentage of good cosmetic and functional results; but rather a higher percentage of fair results. One should realize, that in occipito-frontalis muscle surgery, a balance between mild blepharoptosis and some lagophthalmos must be achieved; both of these rather undesirable situations will be more or less compensated for by contraction and relaxation of the occipito-frontalis muscle.

Utilization of the occipito-frontalis muscle can never do more than suspend the blepharoptotic lid from the occipito-frontalis muscle by sutures, cicatricial bands, skin, orbicularis oculi fibers, or fascia lata strips. Elevation of the lid then can only be accomplished by elevation of the occipito-frontalis muscle. This will produce an unpleasant facial grimace. The natural lid fold is almost impossible to achieve due to the fact that the pull on the lid is up and somewhat anteriorly, when normally the pull from the levator palebrae superioris is up and back into the orbit. Spaeth has stated that this procedure substitutes lagophthalmos for

blepharoptosis. Thus we see that, theoretically at least, use of the occipito-frontalis muscle is not an ideal procedure. This procedure does have the advantage of being the easiest technically of the three muscles to utilize and perhaps creates the least amount of trauma to the lid. At times it must be utilized as will be brought out later. However, it would seem that one should look to utilization of the other two muscles first in the surgical correction of congenital blepharoptosis.

There are a number of disadvantages and advantages to utilization of the superior rectus muscle in the surgical correction of congenital blepharoptosis. Some of the disadvantages are:

1. Postoperative diplopia and hypotropia in unilateral congenital bleparoptosis from adding an extra burden of lifting the lid to the superior rectus muscle's original job of elevating the globe. This was present in upward gaze in one of the two cases in this series.

2. Lagophthalmos during sleep resulting from Bell's phenomenon, that is, as the globe rolls upward with Bell's phenomenon, it must also carry the upper lid upward, if the lid is attached to the superior rectus muscle as it is in this type procedure.

3. It is most unphysiologic to attempt utilization of this muscle if it is already weak. Berke has reported a modification of the Motais procedure wherein he resected the superior rectus muscle, not cutting off the muscle distal to the resection, but using this part of the superior rectus muscle to reach through the levator and be sutured to the anterior surface of the tarsus. Thus he strengthens the superior rectus muscle for its double function of elevating both the globe and lid. This would appear logical to me.

Some of the advantages of this type procedure are:

1. It may be utilized nicely in bilateral blepharoptosis where unilateral hypotropia and diplopia are less likely to occur.

2. It may be used if there is complete blepharoptosis and no levator function or levator muscle present. Spaeth feels that due to diplopia and hypotropia, superior rectus muscle surgery should be reserved for bilateral blepharoptosis with bilateral good superior rectus muscle function, and in situations wherein binocular single vision cannot be attained.

3. Another advantage to this type procedure is that one gets a nice lid fold due to the upward and posterior pull of the superior rectus muscle in elevating the lid. As was pointed out previously the origin and course of this muscle are in close association to that of the levator palpebrae superioris.

4. A fourth advantage is that the lid moves with the globe.

The best cosmetic and functional results and the fewest undesirable complications will be achieved through the judicious use of the levator palpebrae superioris muscle. It seems to me that the main disadvantage with this type procedure is that it cannot be wisely utilized in all cases of congenital blepharoptosis. Some of the advantages of this type procedure are:

1. There is no lagophthalmos with sleep, unless too much levator resection is done.

2. The lid follows the movements of the globe.

3. There is no peculiar facial grimace.

4. A nice lid fold is attained.

5. There is no hypotropia or diplopia resulting from this type surgery.

6. The levator resection may be repeated if inadequate correction is attained on the first procedure.

7. It may be utilized for mild or severe cases of congenital blepharoptosis by resecting less or resecting more levator palpebrae superioris, the range here being from 8.0 to 10 mm. for very mild blepharoptosis up to 16 to 18 mm. for a large amount of blepharoptosis.

To achieve these good cosmetic and functional results with levator palpebrae superioris muscle surgery, one must select the

proper cases in which to use this type procedure. In order to do this one must be concerned with the anatomy and pathologic physiology involved in the blepharoptosis.

Most authors feel that there must be some levator function present. However, in this series a Blaskovics levator resection and tarsectomy was done on at least two lids in which no levator function could be demonstrated. The postoperative results were good cosmetically and functionally. I feel that it may not always be possible to ascertain levator muscle function even with careful examination, although some may be present. However, if the blepharoptosis is due to peripheral mesenchymal aplasia of the levator muscle itself or an extremely thin, atrophic and weak muscle, levator muscle surgery could not be expected to be successful.

One must not resect too much levator as this obviously would lead to lagophthalmos. A sufficient amount of muscle must be resected. This is determined by the number of mm. of needed lid elevation, the size, thickness and development of the levator palpebrae superioris muscle, and the amount of fibrosis of the muscle. Spaeth has set forth a working rule of approximately two mm. of levator resection for one mm. of desired correction. An important part of a levator resection is the tarsectomy. Approximately one half of the vertical width of the tarsus should be excised for two purposes: (1) to shorten the lid and give an actual advancement of the levator as well as the resection; (2) to provide a raw edge for firm reattachment of the resected levator into the tarsus.

The surgical correction of blepharoptosis associated with blepharophimosis was accomplished in this series with levator resection and tarsectomy. One usually finds a rather stiff, fibrous lid which would seem to me to create a burden for the superior rectus muscle to elevate. Resection of the stronger, fibrotic levator should give a more permanent elevation of the rather rigid lid. The occipitofrontalis muscle could be utilized, however less advantageously due to the previously demonstrated faults of this procedure.

The Marcus-Gunn jaw-winking reflex cases in this series were corrected according to the method first developed by Dr. Spaeth. There must be misdirection of the fifth cranial nerve fibers into the levator. First one must abolish the efferent arm of this reflex by section of the levator palpebrae superioris muscle from the tarsus. One now has two muscles left to utilize in the correction of the complete (acquired from section of the levator from the tarsus) blepharoptosis. Since this syndrome is usually unilateral one should utilize the occipito-frontalis muscle to correct the blepharoptosis. Superior rectus muscle surgery may lead to postoperative hypotropia and diplopia.

Pseudo-Graefe phenomenon is a situation of misdirection of regenerating third cranial nerve fibers. Here the levator must be sectioned, forming a complete paralytic blepharoptosis. This paralytic blepharoptosis is then best corrected by utilization of occipitofrontalis muscle.

Duane's retraction syndrome is not a true blepharoptosis, but rather an apparent blepharoptosis due to retraction of the globe into the orbit (enophthalmos) from the pull of the medial rectus muscle against a fibrotic lateral rectus muscle with hypertrophic check ligaments. Thus this entity will not be further considered in this thesis.

Surgical correction of acquired blepharoptosis

Depending upon the etiology, the correction of acquired blepharoptosis may be more difficult technically, but perhaps the correction in relationship to the anatomy and pathologic physiology is somewhat more simple than that for congenital blepharoptosis. The first and most important point to consider is to be certain that the correct diagnosis is known and that the etiologic factor causing the acquired blepharoptosis is stable.

If the levator has been sectioned, it must be found and resutured. If the blepharoptosis is due to scars and cicatrices, these

must be resected and the lid resutured in its anatomic planes, with suture lines placed if possible to prevent notching, reformation of cicatrices and unsightly deforming scars. If it is impossible to resect scars and resuture the areas, then skin grafts and/or mucus membrane grafts must be utilized. Foreign bodies or space-taking lesions must be removed. Atonic blepharoptosis may be corrected by levator resection with tarsectomy and resection of excess skin in cases of blepharochalasis. Blepharoptosis of metabolic disturbances and hysterical blepharoptosis have no surgical component. The correction of blepharoptosis associated with metabolic disturbances is medical and the correction of hysterical blepharoptosis is aimed at correction of the neuropsychiatric disorder. One may be justified in ordering some form of crutch glasses to elevate the lids temporarily in hysterical situations.

Progressive muscular dystrophy is another form of acquired blepharoptosis which is managed safest perhaps with crutch glasses. In this series, however, there were two lids upon which a Blaskovics levator resection and tarsectomy were done with good cosmetic and functional results. The permanency of this correction will depend upon the rapidity of the progression of this disease process. However, this result remains good eight months after surgery.

Conclusions

I feel that if one carefully examines and evaluates each case of blepharoptosis preoperatively, one will see that blepharoptosis is a complicated situation, more so in some cases, less in others. One should be able to predict in advance the extent to which one would expect to achieve previously stated ideal goals; that is, a perfect cosmetic and functional result. One will be able in advance to realize that this is impossible in every case of blepharoptosis.

The result of this investigation leads to the conclusion that utilization of occipitofrontalis muscle results in poorest permanent cosmetic and functional results, although perhaps it is technically the easiest procedure to perform and may at times be the only procedure to be rationally used; that is, with no levator function, complete blepharoptosis and paretic superior rectus muscle. Study of the literature has revealed that utilization of the superior rectus muscle is fraught with the most serious complications of lagophthalmos in sleep and hypotropia and diplopia. The final conclusion is that utilization of the levator, if possible and indicated, that is, with some levator muscle function present, will lead one to the best cosmetic and functional results in the surgical correction of blepharoptosis.

934 Exchange Building.

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Ophthalmic Miniature

Down, England; Mar. 18, 1871

To Sir William Bowman:

When a man coughs, retches or laughs violently tears come into the eyes. Now, in all these cases I observe that the orbicularis muscle is more or less spastically contracted, as also in the crying of a child. Hence I am led to conclude that there must be some relation between the contraction of this muscle and the secretion of tears. Does the same nerve which runs to the orbicularis send off fibers to the lachrymal glands?

Charles Darwin.

NOTES, CASES, INSTRUMENTS

GLAUCOMA ASSOCIATED WITH STEROID THERAPY AND ATOPIC DERMATITIS

JONATHAN L. HARRIS, M.D.
East Orange, New Jersey

A report by Covell has suggested that glaucoma may be induced by prolonged therapy with corticosteroids.[1] Laval and Collier have described a rise in intraocular pressure associated with the use of ACTH.[2] Studies have been conducted to evaluate whether these medications do influence intraocular pressure. A recent survey by Lee concluded that there was no significant tendency to glaucoma or impaired facility of outflow in a group of 13 asthmatics who had been given steroids over a long period.[3] Another study by Tillett showed no pressure rise in a series of patients with normal tension given ACTH.[4]

Present-day treatment of atopic dermatitis has been based largely on systemic and local use of corticosteroids and injection of ACTH.[5,6] Eye involvement in atopic dermatitis is a prominent feature of this disorder. A most serious complication is a specific type of anterior polar cataract. Brunsting, Reed, and Bair have reported this complication as occurring in five to 10 percent of all cases.[7,8] In addition, keratoconus and keratoconjunctivitis are frequently found.[9] Glaucoma has not been described as a specific complication.

The following case report deals with a patient with atopic dermatitis treated by corticosteroids and ACTH for many years, who developed severe glaucoma.

CASE REPORT

A 37-year-old white man was admitted as an emergency case to the eye section of the surgical service of the Veterans Administration Hospital on September 22, 1958, with a diagnosis of bilateral glaucoma. He had been seen by his ophthalmologist on the morning of admission, and had been found to have bilateral elevation of intraocular pressure. His presenting symptoms were ocular pain and blurred vision, worse on the right.

There was a past history of headache and mild visual disturbance for several months. A sudden intensification of symptoms brought him to his ophthalmologist. He had previously been examined periodically for myopia, and a few months prior to his admission had shown no evidence of glaucoma.

A prominent feature of his past medical history included the diagnosis of atopic dermatitis. This skin disorder began while he was in active military service 14 years prior to admission, at the age of 23 years. At that time he suffered a fungus infection of the skin, which was followed by atopic dermatitis. He had received corticosteroids of various types orally and topically for the past seven years. At regular intervals he also was given injections of ACTH. At the time of his admission he was receiving triamcinolone (Aristocort®), 4.0 to 8.0 mg. daily, and ACTH, 20 units every other day. He was under the regular observation of his dermatologist.

The chief findings of the examination on admission showed corrected vision on the right of 20/60 and on the left of 20/30. His prescription was O.D., −2.0D. sph. ⊃ −0.5D. cyl. ax 170° and O.S., −1.5D. sph. ⊃ −0.5D. cyl. ax. × 160°. The tension of the right eye was 80 mm. Hg and the left 40 mm. Hg (Schiøtz). Slitlamp examination showed edema of the corneal stroma on the right. The pupils were constricted by miotics and the fundi could not be well seen. The skin of the face, head, trunk, and extremities showed findings consistent with atopic dermatitis.

Initial treatment consisted of frequent instillation of Pilocarpine (two percent) and eserine (0.25 percent drops). Acetazolamide (Diamox®), 500 mg., was given initially, and then continued in the dosage of 250 mg. every four hours. A profound drop in tension occurred, and 16 hours after admission the intraocular pressure was 10 mm. Hg in each eye. Eserine was discontinued and the strength of the Pilocarpine decreased. Diamox was continued. All systemic corticosteroids were stopped, but ACTH (20 units every two or three days) was ordered to avoid adrenal shock.

Gonioscopy and visual field studies were done following the drop in pressure. Gonioscopy showed bilateral narrow open angles with several anterior peripheral synechias on the right. No synechias were seen on the left. The fields displayed loss of most of the lower nasal quadrant on the right, and no significant impairment on the left. A view of the optic nerveheads showed early cupping and atrophy on the right. The left disc appeared normal.

During the following week the tension in both eyes remained at low normal levels. Pilocarpine (one percent) was given four times daily, and Diamox (125 mg., three times daily). At the end of the week the tension of the right eye suddenly rose to 34 mm. Hg, the left remaining at 17 mm. Hg. During the following six weeks, there was a

constant need to increase the strength and frequency of the Pilocarpine, particularly on the right. Ethoxzolamide (Cardrase®) was substituted for Diamox. Visual field studies done four weeks after the original fields showed no significant changes. Tension of the right eye tended to remain in the low 30's, but at times rose to 40. The tensions on the left remained in the low 20's. An iridencleisis was done on November 19, 1958, eight weeks after the patient was first seen. The postoperative course was uneventful, and characterized by no inflammatory reaction. Local corticosteroid salve (Neo-Cortef®) was used for the operated eye. On November 28th, the ninth postoperative day, the tension of the operated right eye was 6.0 mm. Hg and the unoperated left eye, 23 mm. Hg. Cardrase was discontinued. Pilocarpine (one percent) was used four times daily in the left eye.

On December 1st while at home, the patient took Aristocort (12 mg.) in divided doses because of annoying skin symptoms. On December 2nd the tension was taken and found to have risen to 18 mm. Hg on the right and 40 mm. Hg on the left. He was still using pilocarpine (one percent, four times daily) in the left eye. Unfortunately it cannot be determined whether the cessation of the Cardrase or the administration of the Aristocort were involved in this rise in tension.

Aristocort was prohibited and Cardrase resumed. On December 3rd the tension dropped to 12 mm. Hg on the right and 26 mm. Hg on the left. His medications consisted of Cardrase (125 mg. three times daily) and pilocarpine (one percent to the left eye four times daily). Within a few days the pressure of the unoperated left eye rose into the 30's and then the 40's. Stronger pilocarpine tended to reduce pressure, but caused annoying visual disturbances. In view of the visual symptoms with the use of stronger miotics, it was decided that surgery should also be undertaken for the left eye. Iridencleisis was done on the left eye on December 17th, four weeks after the operation on the right. Again the postoperative course was entirely uneventful and inflammatory reaction absent.

No miotics were used in either eye following the second operation. Carbonic anhydrase inhibitors were also stopped. On December 22nd and 24th, tension in the right eye was 17 mm. Hg and 15 mm. Hg, respectively, and on December 29th, R.E., 10 mm. Hg; L.E., 5.0 mm. Hg.

On January 1, 1959, the patient began to use small amounts of Aristocort—2.0 to 6.0 mg. daily between January 1st and 4th. On January 5th the tension of the right eye had risen to 20 mm. Hg and the left eye to 21 mm. Hg. Aristocort was stopped and on January 7th, tension dropped to 10 mm. Hg on the right and 15 mm. Hg on the left, and on January 9th to 6.0 mm. Hg on the right and 12 mm. Hg on the left. It should be emphasized that no carbonic anhydrase inhibitor had been used since December 17th.

When the patient was seen for follow-up visits on February 10th and March 24th, the tension of the right eye was 8.0 mm. Hg and the left 12 mm. Hg. He was taking 2.0 to 6.0 mg. of Aristocort daily. His vision was corrected to 20/20 in each eye. The filtering operations appeared to be working well.

Discussion

This 37-year-old white man had had atopic dermatitis for 14 years. According to accepted dermatologic therapy, he had received corticosteroids systemically and locally, and injections of ACTH for seven years. He developed glaucoma with an apparent sudden onset at a relatively early age. His glaucoma was severe and resistant to medical treatment.

Filtering operations were done on both eyes. The postoperative course was characterized by a complete absence of inflammatory reaction and by satisfactory healing.

There was no family history of glaucoma, and it seems reasonable to suspect some connection between his glaucoma, his skin disorder, and possibly his treatment with steroids. In the postoperative period there were two episodes of a rise in intraocular pressure following the use of oral steroid. These episodes could not be considered conclusive, but were suggestive, particularly in the second instance. The use of Aristocort several months after the operations, when both filtering tracts appeared well established, did not cause a rise in tension.

Conclusion

1. Further clinical and laboratory studies should be done to investigate the relationship between intraocular pressure and corticosteroid administration.

2. The possibility that glaucoma is yet another ocular complication of atopic dermatitis should be considered.

Veterans Administration Hospital.

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METASTATIC PNEUMOCOCCIC ENDOPHTHALMITIS

Secondary to pneumococcic meningitis

Ernesto M. Almeda, M.D.
New York

Metastatic pneumococcic endophthalmitis is not infrequent; however, the incidence of unilateral metastatic pneumococcic endophthalmitis secondary to pneumococcic meningitis—the case here reported—is rare. Axenfeld[1] was the first to describe a similar case.

Report of a case

History. E. P., a married Puerto Rican woman, aged 30 years, was admitted to St. Luke's Hospital on October 29, 1956, with the complaints of severe headache, stiffness of the neck, chills, and a fever of 42 hours' duration. Six days prior to admission, she had a sore throat that was treated by her private physician.

Physical examination. On admission, temperature was 105°F.; pulse, 78 per minute; respiration, 24 per minute; blood pressure, 150/80 mm. Hg.

The pertinent physical findings were nuchal rigidity; ear, nose and throat, not remarkable; heart, lungs, and abdomen, normal. The deep tendon reflexes were active and equal; the Kernig sign was positive; the Babinski and Brudzinski signs were negative.

Eye examination. In the right eye, there was slight circumcorneal injection. The cornea was clear. The pupil was constricted, and reacted sluggishly to light. The media were clear and the fundus showed no remarkable pathologic changes.

The left eye was essentially negative.

The tension in both eyes was 17 mm. Hg (Schiøtz). No visual acuity test was done because the patient was in a semicomatose condition.

Laboratory findings. Urinalysis showed albumin (4+). The blood showed a hemoglobin of 11.4, a white blood count of 28,000, a polys of 76. Serology was negative. Blood chemistry showed: BUN 13.4, FBS 85, ESR 36 mm. The blood cultures were negative for pathogens. The spinal fluid showed 3,200 cells per cu. mm.; 2,125 wbc.; 90 percent polys; protein, 107 mg./100cc.; sugar, 11 mg./100cc.; chlorides, 117 mg./100cc. The spinal fluid cultures were positive for pneumococcus type XIX. The eye smears and cultures were negative. All the chest X-ray films were not remarkable.

Hospital course. The patient was comatose one and a half hours after admission. She was immediately given massive doses of penicillin intramuscularly and intrathecally, and sulfadiazine, intramuscularly. After 48 hours, her right eye showed moderate circumcorneal injection, with marked clouding of the aqueous. The pupil was regular, slightly dilated, and did not react to light. The vitreous was very hazy and the fundus could not be visualized. Tension was soft by the finger. Perception of light was poor. The left eye was essentially negative.

A diagnosis of metastatic pneumococcic endophthalmitis of the right eye was made. The treatment consisted of atropine, warm compresses and hydrocortisone acetate.

Subsequent examination of the right eye did not show any remarkable improvement. On the fourth hospital day, the right eye was markedly congested; the cornea was hazy, with some grayish deposits on the endothelium; the aqueous was very cloudy; the pupil was dilated, with no reaction to light; and a well-formed vitreous abscess could be seen. There was complete absence of light

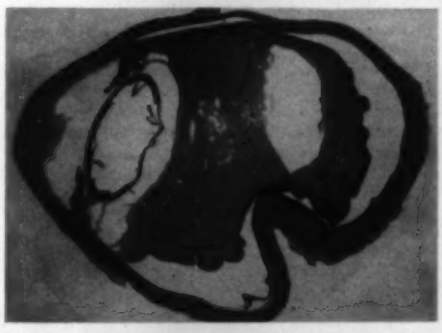

Fig. 1 (Almeda). Numerous inflammatory cells in the anterior and posterior parts of the vitreous. Note vitreous-strand formation.

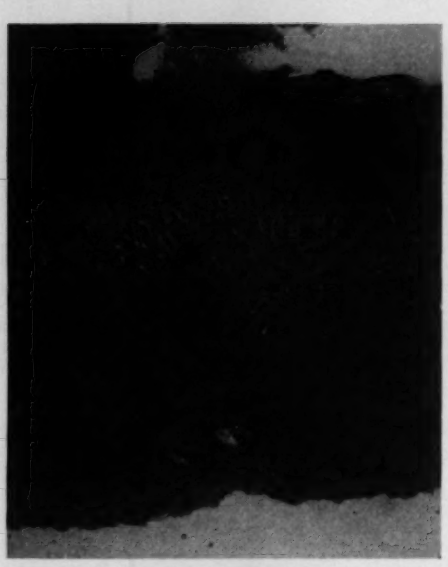

Fig. 2 (Almeda). Perivascular infiltration of the blood vessels in the optic nerve. No bacteria present in the arachnoid spaces.

perception. The eye remained congested without any levation of tension.

On the 31st day of hospitalization, when the patient was clinically improved and her right eye less congested, enucleation was performed.

The patient was discharged as improved, with residual paralysis of her lower extremities and a prosthesis for her right eye.

*Gross examination of the globe.** The globe was normal in size but collapsed.

Microscopic examination.† The cornea was normal, except for many keratic precipitates on the endothelium. The keratic precipitates were made up mostly of polymorphonuclear cells. The limbus was infiltrated with round cells and with a few polymorphonuclear cells. The blood vessels were congested.

In the aqueous, there were albuminous coagulum and numerous polymorphonuclear cells. The angle was open and there were inflammatory cells in the trabecular meshwork. The anterior surface of the iris, the anterior lens capsule and the ciliary body were diffusedly infiltrated with inflammatory cells. The lens showed some cortical opacity. The iris and ciliary blood vessels were congested. In the anterior and posterior parts of the vitreous, there were numerous round cells and polymorphonuclear cells; there was, also, a marked increase of albumin; and, in the posterior part, there were some vitreous strand formations. The pigment epithelium of the pars plana was organized and proliferated in the form of a cyclitic membrane. The inner layer of the retina was markedly infiltrated with polymorphonuclear cells. The choriocapillaris of the choroid showed mild and diffused infiltration of polymorphonuclear cells and round cells.

There were numerous perivascular infiltrations of the blood vessels in the optic nerve. The arachnoid spaces showed proliferation of the endothelium, and round cells were present.

Histologic diagnosis. Endophthalmitis, right eye.

Comment

It is difficult to ascertain the primary focus of infection in this case. However, it can be said generally that, in metastatic pneumococci infections, the intraocular structures can be involved in any of the following ways: (1) hematogenous route (septic embolus and toxins, either through the retinal or uveal system of the blood vessels[2]); and (2) direct bacterial infiltration along the arachnoid spaces into the optic nerve and into the eyeball.[1]

From the available sections, it is evident that there was no direct bacterial infiltration along the arachnoid spaces into the optic nerve and into the eyeball. The intraocular structures were involved via the hematogenous route. The mechanism of infection in this case is utterly different from that of a similar case reported by Axenfeld[1] in 1894.

Summary

A case of fulminating unilateral metastatic pneumococcic endophthalmitis occurring during pneumococcic meningitis is presented. Despite the intensive and massive chemotherapeutic and antibiotic treatment, the eye was lost.

210 East 64th Street (21).

* From the Department of Ophthalmology, St. Luke's Hospital.

† C. C. Teng, M.D., 210 East, 64th Street, New York, New York.

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THE WOLFE SCLERAL TONOMETER

WILLIAM H. HAVENER, M.D., AND JAMES PATTERSON, M.D.
Columbus, Ohio

The Wolfe scleral tonometer was designed by optometrists to measure intraocular pressure without the aid of topical anesthetics. This is a spring-scale instrument activated by a plunger emerging through a hole in the center of a footplate. It is quite crudely constructed in comparison to a modern Schiøtz-type tonometer. Standardization is supposedly accomplished simply by adjusting the plunger to obtain a reading of 70 on the test plate. The Wolfe tonometer may be used in either horizontal or vertical position by varying the pressure of application (as measured by a spring-scale on the shaft of the tonometer).

If the Wolfe tonometer is a reliable instrument, it would be most useful to the general medical practitioner. If not, optometric statements to the effect that "glaucoma has been ruled out" represent false and dangerous reassurance to the unfortunate patient. Since this is obviously an important question, the accuracy of Wolfe scleral tonometry has been evaluated. Seven separate Wolfe tonometers were purchased. They were standardized and used in accordance with the manufacturer's instructions. Five board-certified ophthalmologists and several members of the resident staff independently evaluated these tonometers, checking against Schiøtz values.

Patients found to have elevated tensions with the Schiøtz tonometer were preferentially selected in order to determine the accuracy of the scleral tonometer in detecting known glaucoma. Since scleral tonometry was done in most cases immediately following corneal tonometry, most patients were topically anesthetic at the time of measurement. Without such anesthesia, scleral tonometry was painful, and frequently was not tolerated by the patient.

RESULTS

One hundred and ten patients (219 eyes and one prosthesis) were measured. Seventy eyes (table 1) measured above 25 mm./Hg (Schiøtz) and of these only 13 were detected as glaucomatous by the Wolfe to-

TABLE 1

PAIRED SCHIØTZ-WOLFE TONOMETER READINGS ON EYES WITH SCHIØTZ PRESSURES 25 MM.HG OR HIGHER (ARRANGED IN ORDER OF SCHIØTZ VALUE)

Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe
25	16	28	16	34	10
25	16	28	15	34	13
25	14	28	15	34	13
25	12	28	17	35	18
26	17	29	*33*	36	18
26	18	30	22	36	20
26	20	30	17	42	20
26	16	30	*27*	42	22
26	21	30	*27*	42	22
26	22	30	22	42	*25*
27	15	30	15	42	22
27	17	30	16	46	13
27	16	31	15	46	17
27	14	31	*28*	46	22
27	14	31	16	50	*38*
27	19	31	22	50	*26*
27	*29*	31	1	60	*30*
27	19	31	21	60	20
27	16	32	*27*	60	15
28	*26*	32	*27*	80	2
28	18	32	16	80	18
28	18	33	15	85	17
28	*30*	34	13		
28	15	34	19		

Grateful acknowledgement is made to Drs. James Andrew, Jack Bontley, Robert Magnuson, Torrence Makley, and Harry Sage for their help in this study.

TABLE 2

PAIRED SCHIØTZ-WOLFE TONOMETER READINGS ON EYES WITH WOLFE PRESSURES 25 MM.HG OR HIGHER (ARRANGED IN ORDER OF WOLFE VALUE)

Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe
20	25	14	27
20	25	17	28
13	25	23	29
13	25	16	29
12	25	17	30
17	25	12	30
12	25	15	30
16	25	24	30
14	25	20	30
14	25	24	32
20	25	24	35
21	26		

TABLE 3

PAIRED TONOMETER READINGS BY TWO DIFFERENT WOLFE TONOMETERS

Wolfe I (mm.Hg)	Wolfe II	Wolfe I (mm.Hg)	Wolfe II
15	11	8	10
12	9	12	12
18	13	10	16
14	11	12	15
26	15	16	12
13	14	14	11
15	13	22	18
16	10	25	19
38	22	20	22
26	24	18	15
18	15	18	20
20	23	30	21

nometer (57 of 70, or 82 percent were false negative). Twenty-three eyes (table 2) were false positives (normal Schiøtz, elevated Wolfe), representing 15 percent of the 149 eyes with normal pressure. The average difference between the Schiøtz and Wolfe readings was determined by adding all the individual differences and dividing by the total number of eyes (219). The average age difference between Schiøtz and Wolfe values was 8.4 mm. Hg. Each of two Wolfe

TABLE 4

PAIRED SCHIØTZ-WOLFE TONOMETER READINGS ON EYES WITH PRESSURES BELOW 25 MM.HG BY BOTH

Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe	Schiøtz (mm.Hg)	Wolfe
20	12	17	18	22	19	24	19
20	12	16	14	17	15	15	15
13	12	15	14	17	17	17	17
13	12	17	14	18	13	17	17
15	11	16	14	18	12	18	12
15	11	17	14	19	21	16	23
20	15	15	15	19	15	21	17
17	15	17	15	22	20	20	15
20	11	19	15	15	18	20	10
17	15	17	15	18	20	17	20
20	11	15	15	9	15	24	15
23	12	15	14	24	19	20	15
23	12	14	16	22	17	24	18
23	12	14	16	23	11	22	19
23	13	17	22	24	20	16	12
14	14	19	16	24	19	17	14
14	12	19	18	24	17	20	19
12	6	17	15	23	15	19	16
14	8	17	15	21	12	17	18
23	13	17	22	16	18	23	15
20	14	17	22	20	14	20	21
12	13	13	16	12	13	10	23
12	16	19	18	24	15	16	21
15	15	14	16	16	18	22	23
20	22	14	18	17	8	22	22
15	15	16	18	19	12	23	19
15	14	17	18	17	10	23	14
17	22	17	18	19	12	23	16
19	18	22	16	16	16	22	12
17	18	22	19	16	14	24	14
19	21	16	17	23	14	23	11
				22	17	21	9

TABLE 5

WOLFE TONOMETER

False negative—(82%)—57 of 70
False positive—(15%)—23 of 149

tonometers was used to measure 24 eyes (table 3). The average difference between these two Wolfe tonometers was 4.3 mm. Hg.

DISCUSSION

It was our impression that the Wolfe tonometer is not sufficiently reliable to be of any clinical use. False reassurance based on this technique of scleral tonometry will delay detection of 82 percent of patients with glaucoma.

SUMMARY

In this study the Wolfe scleral tonometer detected only 14 of 70 eyes with elevated tension. The average difference between Schiøtz and Wolfe readings was 8.45 mm. Hg. The Wolfe tonometer cannot be recommended for clinical use.

Ohio State University (10).

ALPHA CHYMOTRYPSIN AND RUPTURED HYALOID

LEONARD FLOM, M.D.
Fairfield, Connecticut

Following is a report of the use of Quimotrase[1, 2] in the presence of known rupture of the hyaloid membrane:

CASE REPORT

A 39-year-old white man, a machinist, was first seen the day following an accident in his shop. He felt a foreign body strike his right eye but thought nothing of it until the next day when the eye remained persistently irritated, inflamed and tearing.

Examination revealed the cornea had been perforated at the 9-o'clock meridian approximately three mm. from the limbus. The lens was perforated anteriorly in the same meridian and posteriorly slightly temporal to the optical center. As described many times, the posterior perforation was three or four times larger than the anterior one. This is often seen in gunshot wounds elsewhere on the body.

Fundus examination with the Schepens ophthalmoscope revealed a glistening metallic object embedded in the retina superiorly and slightly temporal to the macula. The retina surrounding the metallic object was elevated and detached and a large hemorrhage was seen directly inferior to this. Field examination was unsatisfactory because of lacrimation and inability to fixate steadily. Vision was 20/50 and tension 15.5 mm. Hg (Schiøtz) with the 7.5-gm. weight. X-ray localization confirmed the metallic foreign body, placing it very close to the macula (as was seen in fundus examination).

The foreign body was visualized in the operating room with the Schepens ophthalmoscope, and removed with the aid of an incision posteriorly through which the electromagnet was inserted. The area surrounding the incision was touched with the ball diathermy. This was done in October, 1958. Following this the retina healed nicely, but the cataract rapidly matured until the patient had only light projection. Before complete opacification had occurred, it was noted that the posterior lens capsule was adherent to the vitreous in the area surrounding the rupture.

In January, 1959, three months after the accident, the surgery was performed. Anesthesia was Xylocaine (one percent) and Wyadase. Van Lint akinesia and a retrobulbar injection of two percent Xylocaine with four minutes of massage were used. A limbal-based flap and two preplaced sutures were used. Two iridotomies were made in the iris and alpha chymotrypsin was inserted under the iris inferiorly and superiorly through the iridotomies. The 1:5000 dilution of Barraquer's Quimotrase[1] was used. It was left in the anterior chamber two minutes. The Bell erisophake was applied, making certain that the suction was applied over the rent in the anterior lens capsule. Pressure was applied from below with the muscle hook. The lens was delivered extracapsularly and the cortex and nucleus seemed to shell right out of the capsule. The capsule was then grasped with the Schweiger forceps and easily removed. There seemed to be no lens remnants in the anterior chamber and this was later confirmed by slitlamp examination.

One more postplaced suture was inserted and air was placed in the anterior chamber. The postoperative course was uneventful and the patient was discharged on the fifth postoperative day. His vision is correctible to 20/20+ in that eye and he is now being fitted with a contact lens. The slitlamp examination showed the perforated areas in the hyaloid to be slightly irregular but the rest of the membrane appeared to be intact. The anterior chamber was perfectly clear and the tension normal. The healed retinal area gave the appearance of a healed choroiditis with much pigmentation.

COMMENT

In retrospect, I think the lens delivered extracapsularly because of the adherence between the posterior capsule and the hyaloid. In spite of the fact that this was a 39-

year-old man, the lens was delivered with a capsule adherent to the vitreous; however, no remnant whatsoever remained in the anterior chamber. It would certainly seem probable that the zonulolysis[1] was responsible for the ease with which the capsule was extracted. Even though the vitreous face was ruptured and was in contact with the enzyme, no pathologic change in the vitreous has occurred to date.

SUMMARY

A case is reported in which alpha chymotrypsin was used in the presence of a ruptured hyaloid to extract a lens in a relatively young man. There were no adverse affects. The resulting vision was excellent, and I feel the extraction was made far easier by use of the enzyme.

70 Reef Road.

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JUVENILE DIABETES

ISADORE GIVNER, M.D.
New York

The term "juvenile diabetes" is reserved for those patients whose diabetes begins before the age of 15 years. Recent studies by Kornerup[1] have shown that diabetes began before the age of 15 years in 57 percent of those with proliferative retinal changes. In the series by Root, et al.,[2] the incidence was 42 percent. These are high percentages considering that only 13 percent of all diabetic persons have the onset of the disease during the first two decades. The average duration of the diabetes in this group before the development of proliferative retinopathy was 17 years.

At camp NYDA, sponsored by the New York Diabetic Association for Diabetic Children, an opportunity of studying 1,000 eyes of juvenile diabetes was had over a period of 12 years (1946-1958). Cohn studied the beta-lipo proteins in 600 diabetic children. Lax carried out vasculograms on juvenile diabetes to study early evidence of changes in tonus in the arterial wall. In none of these children was arteriosclerosis observed ophthalmoscopically. The changes detailed in the vasculograms are also found in arteriosclerosis, yet one cannot say that these changes exclude mere rigidity as opposed to suppleness, for we do not know what molecular changes are responsible for these peripheral changes.

All children were studied under a mydriatic (Paredrine, one percent). Pinpoint hemorrhages or microaneurysmal dilations were found in one percent of these children; 0.4 percent had lenticular changes (table 1).

Cohn found 54 percent to have elevated beta-lipo proteins in contrast with controls in New York City of 10 percent having elevated beta-lipo proteins. Seventy of the diabetic children were followed five years. Cohn believes a vascular toxin related to fats in the diet may play a part in the changes in the veins and capillaries. He suggests that the fat intake should be kept to 30 to 40 gm., with the addition to the diet of unsaturated fatty acids.

In a personal communication Lax reports "In our series we examined 162 diabetic patients between the ages of 11 to 29 years. None exhibited any clinical evidence of arteriosclerosis or hypertension. A control series of 240 presumably healthy young individuals of the same age group were examined. Sixty-two percent of our diabetic patients had abnormal arterial pulse waves. In contrast, only eight percent of the normal group had abnormal pulse wave recordings. The tracings that were abnormal show either diminished or absent dicrotic waves."

TABLE 1

FINDINGS IN THREE SURVEYS OF JUVENILE DIABETICS (1946–1952–1958)

Age (yr.)	Visible Fundi		Hemorrhages + Microaneurysms		% Fundi		Cataracts		% Lenses	
	NYDA	Waite & Beetham	NYDA	Waite & Beetham	NYDA	Waite & Beetham	NYDA	Waite & Beetham	NYDA	Waite & Beetham
Under 10	318	130	None	None	1%	0.6%	4	0	0.4%	0
10–21	682	464	10	4						

It is interesting that no diabetic retinopathy has been reported in a child younger than 10 years of age. It is also a fact that no abnormal pulse tracing has been reported under 10 years of age.

The vasculograph converts pressure waves into electrical energy. The electrical impulses are then amplified and recorded. A sensitive rubber cuff with an inelastic backing is applied to the external surface of the digit to be tested. The cuff is connected by a system of tubing to a diaphragm mechanically linked to a piezo electric crystal. By this means, the crystal transforms pressure changes in the cuff, due to arterial pulsations, into changes in electrical potential. These changes are amplified and activate a string-type mirror galvanometer and are recorded on standard 12-cm. wide electroencephalogram paper. The vertical lines (fig. 1) are time signals 0.1 of a second apart, and the horizontal lines represent pressure increments of 10 mm. Hg in the sensitive rubber cuff applied about the finger. The dark black line represents the applied cuff pressure. The dicrotic wave "D" is absent in the presence of arteriosclerosis.

The pressure within the cuff can be elevated to any desired level. Arterial pulsations appear to be greatest when the applied pressure approximates the intra-arterial diastolic pressure. A complete set of tracings can be taken in 15 to 20 minutes.

A normal vasculogram shows an initial wave and a well-defined dicrotic wave. Consistently in arteriosclerosis, the dicrotic wave

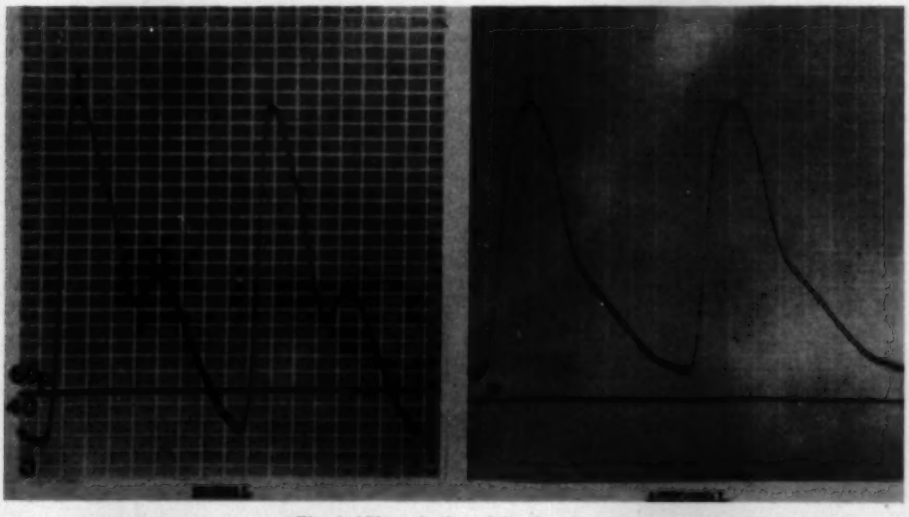

Fig. 1 (Givner). Arterial pulse wave.

Fig. 2 (Givner). Fundus in a case of proliferative retinopathy after long-term testosterone therapy.

is diminished or absent. The dicrotic peak is a reflected wave initiated by the recoil of the arterial blood column against the closed aortic valve. Increased "tonus" of the vascular wall may abolish the dicrotic wave such as in hypertensive vascular disease.

Lax found duration of diabetes to play a part in abolition of the dicrotic wave. Under five years' duration, 51 percent showed abnormal changes. Over five years, 76 percent showed abnormal changes.

A reduction in adrenal cortical function seems to leave a favorable effect on diabetic retinopathy. Pituitary necrosis, adrenalectomy, and the administration of testosterone, the latter through inhibition of the pituitary, have been shown to be of benefit in slowing down and even stabilizing a retinal pathologic alteration. Infection and the administration of ACTH have speeded up the development of diabetic retinopathy.

Saskin and his co-workers[3] suggested the use of testosterone in the treatment of diabetic retinopathy. Patients with well-developed proliferative retinopathy and hemorrhages have shown stabilization of these conditions after intramuscular administration of 25 mg. of testosterone every five to seven days.

A favorable case was that of B. Z., a girl, who was first seen June 24, 1955. Vision was: R.E., 4/200; L.E., 20/100. The left eye showed proliferative retinopathy with fresh vitreous and preretinal hemorrhages. In addition to regulation of her diet and administering medication to reduce capillary fragility, testosterone was administered. To date, she has had over 100 injections with no evidence of masculinization. The vitreous is clear, the fundus free of hemorrhages or exudates, and her vision is 20/20 (fig. 2). She is back at full-time work as secretary.

What else can be done to help these unfortunate patients? Those patients with diseased vessels frequently show hemorrhage after hypoglycemic reactions. Every effort should be made to avoid this by careful observation, diet, and regulated exercise. In the literature some excellent results have been reported with the use of Kempner's rice diet. If one can get a patient to adhere to this diet, it is certainly worth a trial.

One case that may not be rare gives food for thought.

A. B., a woman aged 50 years, has had diabetes since the age of 15 years, at which time insulin was started. Today, 35 years later, her blood vessels are normal and there is only one microaneurysmal dilation in her right eye and one punctate exudate in her left eye, with vision of 20/20, O.U. In trying to ascertain if there was anything in her history that might be related to her excellent vascular status, it was learned that 11 years after her diabetes started, a denervation of the left adrenal was carried out by the late Dr. Crile to see if her diabetes could be cured. Of course it was not; however, at this time, her 17 keto-steroids are 4.8 mg. (normal 5.0 to 14 mg. for females).

Could it be that reduced function of her adrenal, dependent on either innervational disturbance or incidental adrenal damage, could have had a beneficial effect?

A study of all patients with remissions in diabetic retinopathy, whether it seems spontaneous or related to physical or dietary factors, should be carefully scrutinized in hope that some common denominator shows itself. At the same time, search should be continued for a venous and capillary toxin associated with diabetes that might be neutralized or held in check.

108 East 66th Street (21).

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SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

January 19, 1959

Held jointly with the Section on Ophthalmology of the New York Academy of Medicine

DR. ARTHUR LINKSZ AND DR. CHARLES PERERA, *Co-Chairmen*

PANEL DISCUSSION: OPHTHALMIC PROBLEMS IN MEDICINE AND PEDIATRICS

MODERATORS: DRS. STUART COSGRIFF, ISADORE GIVNER, CONRAD RILEY, HENRY WAGENER, AND GEORGE WISE

DR. PERERA: This evening, we are having a panel discussion, or presentation, if you will, on the subject of pediatric and medical ophthalmology.

Dr. Linksz has suggested that I open the questions, reading the first five questions and hearing the responses from the five panelists and then he will take over the chair.

The first question is directed to Dr. Cosgriff.

What is the role of tranquillizing therapy and of early ambulation in ophthalmic surgery?

DR. COSGRIFF: I think that there is a very real need for emotional support in ophthalmic surgery, as in all kinds of medical and surgical practice, if emotional support is what we mean by tranquillizing therapy, particularly in persons who are to undergo or have recently undergone eye surgery. As an internist, I am rather impressed by the remarkable amount of apprehension on the part of some eye patients, particularly the individual who is to have eye surgery for the first time. This is particularly impressed upon me when a well-controlled diabetic will go out of control and actually develop either ketosis or acidosis. I see this complication several times a year, associated with stress of the emotional type just during or just after surgery, and much more directly due to emotional stress than to dietary factors or insulin dosage violation.

At the risk of appearing to pose as a philosopher, I think that the best tranquillizer is probably the doctor himself who, by his careful and complete explanation, reassurance, and thoughtful attention reassures the patient. The nursing staff can also assist in reassuring the patient.

I think that at times, in our earnest effort to give our patient the most precise, advanced scientific therapy in medical or surgical or ophthalmologic problems, we physicians may lose sight of the fact that the patient and his emotional status are really of tremendous importance. I would think that explanation and reassurance are ways to orient him to the ophthalmic problem and, as such, are most worth while approaches to tranquillization.

Explanation—the diagnosis and proposed therapy, the type of operation and the postoperative course, what the patient is to expect after an operation, and so forth—play, I think, a very real role with my patients in making them more amenable and actually better patients.

An example of an outstanding associated emotional stress is the patient with retinal detachment. In this situation the patient enters rather abruptly upon a very emotionally charged period of his life. He suddenly finds himself with serious visual dfficulties, needing an operation and not knowing a great deal about his particular problem.

One way in which we as physicians could exert a tranquillizing effect would be to permit the patient up and about the hospital for a short period of time prior to the oper-

ation. During this time, he would become accustomed to the hospital, accustomed to the fact that he is going to have an operation, and accustomed to the people with whom he is going to be associated during the next couple of weeks. Postoperatively, early ambulation contributes a good deal to tranquillization and certainly is a physiologic step in the right direction. It has been my experience that patients permitted earlier postoperative ambulation, particularly those who have had retinal detachment or cataract surgery, show less frequent circulatory, respiratory and abdominal complications. There has been a rather remarkable decrease in the incidence of abdominal distention and even in such organic complications as acute cholecystitis. Reduction in complications by way of early mobilization is found not only in eye patients but also in those who have been ill with such medical diseases as coronary occlusion, cardiac failure and so forth. Early mobilization really decreases the occurrence of undesirable muscular and vasomotor changes which take place in a patient who has been in bed and not using his muscles for a week or two weeks. These undesirable changes predispose to such complications as emotional depression, loss of the sense of well being, and thrombo-embolism. I think that early ambulation is a physiologic contribution toward normalcy and is certainly, in its own fashion, a good tranquillizer.

The tranquillizers which are commercially available also make a contribution. I think that the older ones, such as phenobarbital, are still satisfactorily effective and very much to be relied on, as are the more recent agents which are, perhaps, more chemically oriented to the precise problem of tranquillization.

What systemic diseases with ophthalmic significance have you encountered in children in your practice?

DR. GIVNER: Ophthalmologists have not only an opportunity but also a responsibility to uncover systemic disease that might otherwise go unrecognized. Syphilis has been conquered fairly well but is still with us and we should consider it when surveying the etiology of eye diseases. In the last issue of THE AMERICAN JOURNAL OF OPHTHALMOLOGY, Dr. Rucker presented a thousand patients who had 3rd, 4th and 6th nerve paralyses and he concluded that 1.5 percent had syphilis. In a like series of cases about 30 years ago, that would have been 48 percent.

Recently, a seven-year-old girl, came in for a routine checkup. On examining with the slitlamp, I found an infiltration in the deeper layers of the cornea, with vessels that still had blood in them. She had a positive Wassermann and, therefore, I suggested that the mother and father both have Wassermann tests; both came back positive. It was the first intimation to them that they weren't in perfect health.

We all recognize that children should be examined by an ophthalmologist before they enter school. However, a child with squint should be examined as soon as the squint makes itself apparent; at times, we may find that a pathologic condition in the macula is causing the divergent strabismus.

Kaposi's varicelliform dermatitis is a generalized vaccinia or disseminated herpes infection engrafted upon a chronic dermatitis, usually an atopic eczema. In order to determine whether or not it is herpes or vaccinia the base of the vesicles should be examined for a virus type of giant cell. Take one of the vesicles on the skin, wipe it with alcohol, open it, scrape the inside of the vesicle base and put the smear on a slide. Stain it with a Giemsa stain at pH 7.0, then if a virus type of giant cell is found, the infection is herpes, herpes zoster, or chickenpox. This type of cell is not found in vaccinia. With a herpetic infection in the eye, a dendritic keratitis is found. I have seen three cases of this disease.

In one patient, a boy who came in with divergent strabismus, a lesion in the macula of the turned-out eye was Coats' disease or retinitis exudativa. This eye was not enucleated; however, to refresh our memories

on the pathology in Coats', here is a slide which shows the great changes in the vessels —microaneurysmal dilations, and so-called ghost cells.

The skin lesion of tuberous sclerosis frequently first appears in children three or four years of age. Angiofibromas are found on the face. Later, when the child is about seven or eight years of age, the so-called Pringle's disease, or adenoma sebaceum, is found. In the fundus of the eye, there are cysts which, if watched for a number of years, will become impregnated with calcium and take on a so-called mulberrylike appearance. If one sees a child or baby with a history of convulsions who has a cyst of this nature in the fundus, one must rule out tuberous sclerosis. That can be done by taking an encephalogram in which one looks for and probably finds the so-called candle droppings of the ventricle, which are nothing but subependymal tumors penetrating into the ventricle.

Another of the phakomatoses to consider is neurofibromatosis. This patient, two and one-half years of age, was brought in because of unilateral exophthalmos. Papilledema was found on the side of the exophthalmos. At first, the cause was hard to determine. A general examination, however, showed cafeau-lait spots on the back. That indicated neurofibromatosis and the relationship of neurofibromatosis and glioma of the optic nerve was recalled. X-ray examination showed that the optic foramina were markedly enlarged. In the past these cases came to enucleation or removal of the optic nerve. More recently, Taveras at the Neurological Institute has recognized that these are astrogliomas and many of them are sensitive to radiation. He treated this child by radiation and five mm. of exophthalmos disappeared; the child was, however, left with postpapilledemic atrophy.

One of the phakomatoses which occur in children is Sturge-Weber's disease or encephalofacial angiomatosis. Up to this point, I always thought of this as being a lesion which affected the brain and the skin. From the last case which I had an opportunity to see and which came to an autopsy, I learned otherwise. There was an angioma in the choroid and it was found that there were angiomas throughout other parts of the body, for instance, in the brain which in this four-year-old child should have weighed about 1200 gm. and weighed only 800 gm. due to marked atrophy in one of the lobes. He had angiomatosis in the meninges, the cortex, the lung, the thymus, the spleen, the testicles and the pituitary. This would indicate that one is not correct in calling this disease encephalofacial angiomatosis; a better name would be diffuse angiomatosis. If there is an opportunity to do a post-mortem study of such a case, it should be complete to see whether this finding is a common or just an incidental finding.

Reese pointed out some time ago that, in the presence of unilateral or bilateral exophthalmos, there is the possibility that the lesion of the orbit is metastatic from a sympathicoblastoma. This was such a case. Another point that Reese brought out was that, if the ophthalmologist sees a patient with hemorrhages in the lower lid, the lesion may be due to necrosis beginning in a metastatic lesion of the orbit.

In a child, one of the most common abdominal tumors is a kidney lesion. Occasionally, a lesion metastatic to the orbit from a Wilms tumor of the kidney is seen.

Stevens-Johnson disease allows a wide range for preventive ophthalmology. The disease itself may have an allergic, toxic, or virus etiology. The lesions in the eyes show membranous conjunctivitis. Every case that has been cultured and repeated has shown Staphylococcus aureus. This is what causes the secondary infection and the corneal ulceration which, if it is not taken care of, may lead to endophthalmitis. That can be prevented.

The pediatrician should call in the ophthalmologist as soon as a conjunctivitis appears so as to prevent other complications. The advice holds for measles. When the

exanthematous rash appears in measles, both corneas will be studded with hundreds of punctate lesions and it is they which cause the photophobia of which the child complains. The room should be darkened, the patient should be cautioned against rubbing the eyes with the fingers and, if any mild conjunctivitis is present, antibiotics should be used as a preventive. In that way many of the scars due to secondary infection will be prevented.

This little boy came into the clinic at the New York Eye and Ear Infirmary with granulation tissue under the upper lid and enlarged preauricular glands—so-called Parinaud's oculoglandular syndrome. A biopsy of the tissue of this lid showed it to be sarcoid. Here we see epithelioid cells, lymphoid cells, giant cells, but no caseation.

Finally, essential or familial hypercholesterolemia is a disease which may occur at any age and usually is familial.

Discussion. DR. RILEY: I should like to ask Dr. Givner a question. Parinaud's conjunctivitis, in the earlier descriptions, was often associated with ownership of a cat. Is there any relationship between this and what has subsequently been described as cat-scratch disease, which is associated with swollen nodes and is apparently viral in origin?

DR. GIVNER: In the early days, the dermatologist used to call everything eczema until he found out otherwise, and then, gradually, not everything was pulled out of that one box. The same thing applies to Parinaud's ocular-glandular syndrome. Every case showing a lesion on the conjunctiva and enlarged preauricular glands was dumped in that category. Presently syphilis was eliminated from the etiology, then tuberculosis, and finally, the major cause of the Parinaud's ocular-glandular sydrome was found due to leptothricosis, as described by Verhoeff. The Leptothrix is frequently carried by cats. Cat-scratch fever may present the picture of Parinaud's syndrome—conjunctival granulation tissue and associated preauricular gland involvement.

What ocular problems do you encounter in your practice as a pediatrician and how do you handle them?

DR. RILEY: Since Dr. Givner covered such a large area, there is not much for me to say. Pediatricians are reluctant to handle ocular problems by themselves. It is interesting that the pediatrician rather prides himself on being a sort of general practitioner in a special age group and when it comes to ear problems or to genitourinary problems or even orthopedic problems, he feels that he can at least begin the attack on the problem fairly competently. When it comes to ocular problems, however, he feels himself extremely insecure and almost immediately refers his patient to an ophthal mologist.

There are two things, I think, a pediatrician would be willing to deal with himself. First would be a simple conjunctivitis that would respond to an antibiotic ophthalmic ointment. Secondly, when there's a history of trauma that might suggest a corneal abrasion, he might put a patch over the eye for 24 hours. This would often be enough but, if the child doesn't improve in that time, he would send him to an ophthalmologist. For the most part, however, ocular problems are something that a pediatrician immediately refers to an ophthalmologist.

Some of the conditions we look for and which we feel are our responsibility to recognize might be mentioned briefly. I think the most important of these, fortunately now a matter of history rather than a current problem, is retrolental fibroplasia. Occasional cases still occur and we don't quite understand what causes them. Because of the frequent occurrence of this disease 10 years ago, many pediatricians became proficient in the ophthalmoscopic detection of the early signs. This, fortunately, is pretty much of the past.

In routine office practice, when we see our babies for monthly checkups and give them their shots, one of the things we ought to do is to shine a light in the baby's eye and see if a silver reflex or a yellow reflex is present,

suggesting a retinoblastoma. This certainly is one condition in which something constructive can be done if it is recognized early. However, in most instances this is probably first picked up by the mother. I don't think it should be. Another thing, as Dr. Givner showed, is tumor of the orbit with a unilateral exophthalmos. He suggested metastasis from a Wilms tumor, neurofibroma, and, of course, neuroblastoma of the orbit. Something no longer seen around New York City that used to be not too uncommon was hemorrhage into the orbit associated with scurvy. I haven't seen such a case in years and probably will never see any. Exceedingly rare in childhood—I have seen only one case—is detachment of the retina, presumably congenital. Two diseases associated with chorioretinitis recognizable at birth or shortly thereafter have just been mentioned briefly. Toxoplasmosis has been known a good many years but, in recent years, a new systemic disease has been discovered which would appear to be viral in origin. It is transmitted from the mother and is know as cytomegalic inclusion disease. It also may be associated with chorioretinitis, may show calcification intracranially, and, therefore, may be quite easily confused with toxoplasmosis.

Some of the conditions more common in adults, for example, the vascular changes in the eyegrounds associated with renal disease or with hypertension of varying causes, are much more rare in children. I have seen an occasional case of pheochromocytoma with a severe degree of papilledema which has responded after removal of the tumor and reduction of the blood pressure to normal. Renal disease in children is usually so slowly progressive that the retinopathies associated with Bright's disease in the adults are seldom seen. These are just a few of the diseases that may occur. The pediatrician should be alert in diagnosing cases that should be turned over to the ophthalmologist. I suppose the commonest referrals are for strabismus and refractive error. I'm afraid, however, that these conditions are often picked up because the child does poorly in school and not because of intelligent inspection by the pediatrician. As pediatricians, we know little about ophthalmology and the minute we suspect trouble we send our patients to ophthalmologists.

Discussion. DR. PERERA: I'd like to ask Dr. Riley whether he thinks most pediatricians examine their patients' vision as early in life as they can. We know that children with defective vision are often picked up at school. Sometimes a school picks up a child with defective vision and it really isn't because the child just says I can't see the next line—if they are pushed into seeing it, they will. Do you routinely make an examination of the visual acuity?

DR. RILEY: I think that when the child is four, five, or six years of age we are usually forced into doing something about visual acuity by the mother who wants to know whether the child can see all right and we all of us have some rather crude animal objects of different sizes hanging on the wall which I think are imperfect as far as charts go: We ask them whether it is a dog or a house and, when the child scores more poorly than he is supposed to, we are apt to think it is because he doesn't understand. I must say that I don't think we do a very good examination in that age group. Later on, when children know the alphabet or when we can count on them seeing the E and responding intelligently, we do a little better, but it is a pretty crude examination and I'm not very proud of it.

In the light of present day concepts, what classification of hypertension has most clinical validity for the practicing ophthalmologist and medical man?

DR. WAGENER: There is no single classification that is perfect. In order to classify hypertension satisfactorily, there has to be a sort of dual classification. Hypertension has to be classified by type, and it has to be classified by degree. As Pickering states in his recent text, the purpose of any classification is really to point the way to diagnosis and to therapy based on the proper diagnosis. Two

recent classifications are Pickering's classification and the classification of a committee of the A.M.A. of which Dr. George Perera, Dr. Wilkins, and Dr. Brust were members. These classifications are essentially the same except for terminology and for some difference in the concept of the nature of hypertension. I do not know if you are familiar with Pickering's work. Pickering's concept of hypertension at the present time is that it is entirely a quantitative matter, that there is no qualitative difference between elevated blood pressure and normal blood pressure and there is no actual qualitative difference between the various, what might be called, forms or phases of hypertension; there is only a difference in the height of the blood pressure. Most of us, so far, have not quite been willing to accept this idea. The Brust-Perera-Wilkins classification still retains, which I think is good, the suggestion of a possible qualitative difference between the types of hypertension.

You know that the first division is into systolic and diastolic hypertension. Systolic hypertension has no very great significance from the eye standpoint. It is a matter largely of loss of elasticity of the larger vessels. The type of hypertension that is classified in detail is really diastolic hypertension in which the systolic and diastolic pressures are both elevated, but primarily the diastolic. This has to be classified into primary and secondary hypertension. In secondary hypertension, there is a known cause for the elevation of blood pressure. I think the term "primary hypertension" is better than "essential hypertension" for those cases in which no specific cause of the elevated blood pressure can be found. There are a number of known causes for secondary hypertension, such as toxemia of pregnancy, glomerulonephritis, pyelonephritis, unilateral atrophic kidney and tumors of the adrenal, and, as each year goes by, we will probably find additional causes.

It should be recognized that hypertensive changes, arteriolar lesions, the retinopathy and, until submitted to certain pharmocologic tests, the elevation of blood pressure itself, are indistinguishable in the primary and secondary types. In either, as noted in both of these classifications, there can be a slowly progressive or essentially nonprogressive phase or form, which used to be called benign hypertension, and a more serious phase or form, called in the Brust-Perera-Wilkins classification the "accelerated form" and in the Pickering classification, the "malignant phase."

Clay, Gipner and I attempted, several years ago, to make a classification of hypertension which would combine these two features—the degree and the type of the hypertension. That is really not very feasible so I think it is well to retain the dual type of classification. I think the term "established hypertension," which was suggested by the A.M.A. committee, may be a better term than "chronic hypertension." I think the "accelerated form" of hypertension is difficult to understand unless it is subdivided. I still think that the classification which we use at the Mayo Clinic (applicable, however, only to the chronic or established form of hypertension) is a relatively satisfactory one.

It should be admitted that there are certain cases in which the blood pressure is not constantly elevated and it is well to retain the term "intermittent hypertension" for this form and, therefore, recognize an "intermittent" and an "established" hypertension. In the fine print of the text explaining the A.M.A. classification, it is stated that sometimes the accelerated form of hypertension is not superimposed, as it usually is, upon established or chronic hypertensive disease but develops as a primary disease process. For this type of accelerated hypertension, I think we should use the term "acute hypertension" as was suggested in the American Ophthalmological Society classification.

Thus, hypertension might be classified into acute hypertension, either primary or secondary, and established hypertension, either primary or secondary. For greater accuracy in classifying hypertension on the basis of

degree or severity, established hypertension can be subdivided into four rather than into two groups. To avoid all the difficulty with terminology, which is really what has caused most of the trouble with classifications, it would be well to retain simply the terms Group 1, Group 2, Group 3 and Group 4 as used in the Keith-Wagener-Barker classification, which in practice seems to have served the purpose well.

There is a certain validity in separating established hypertension into four groups rather than two groups in that there is a distinct difference in the survival curves of Group 1 and Group 2, which are essentially the nonprogressive types, and also between those of Group 3 and Group 4, which belong to the accelerated phase.

There has always been a certain amount of argument as to which cases to diagnose as malignant hypertension. Practically everybody now agrees that, if the term malignant hypertension is confined to cases that show papilledema, diagnosis and prognosis are more accurate. This criterion does not apply to the entity of malignant nephrosclerosis as considered from a histologic standpoint. From a purely clinical standpoint, however, one is safer in diagnosing malignant hypertension (or the Group 4 subdivision of accelerated hypertension, which is its equivalent) only when papilledema is present.

As you probably know, it is very interesting that, in chronic glomerulonephritis, the ophthalmoscopically visible changes are most often those of the retinopathy of acute hypertension, in which there is a rather marked narrowing of the retinal atrerioles without any evidence of preexisting sclerosis. The retinal findings of established hypertension are not as common in chronic glomerulonephritis as one would think they would be.

In Group 1 hypertension, there is minimal narrowing of the retinal arterioles. In cases diagnosed clinically as Group 2, the arteriolar changes are more advanced. There may be grade 2 sclerosis of the retinal arterioles with arteriolovenous compression. The hypertensive changes in this type are often combined with atherosclerosis, which may alter the prognosis of the case.

The classification from the ophthalmoscopic standpoint may depend on whether you assume that, whenever there are localized constrictions in sclerosed arterioles, you are dealing with a progressive hypertensive disease, or whether you consider the constrictions to be atherosclerotic. Personally, I am inclined to think that most of the constrictions are primarily functional and that such a case should be put into Group 3, if the constrictions appear to be active or recent, even though there is no retinopathy. A definite retinopathy, of course, along with the vascular changes is a Group 3 hypertension, although there is only a localized retinopathy. A rather diffuse type of retinopathy with visible anemia suggests a Group 3 hypertension in chronic glomerulonephritis because of the anemia that is visible. Definite chronic vascular changes indicate a well-established retinopathy of Group 4 hypertension.

Actually, it is not difficult to separate cases into such classifications, even for an internist. The internist will gain a certain amount of benefit by classifying his cases of hypertension a little more in detail than simply into benign and accelerated forms.

Discussion. DR. WISE: Dr. Wagener's classification has certainly stood the test of time and its remarkable simplicity makes it extremely useful. I find it still the most satisfactory classification and personally still use it.

There are two or three points which do warrant discussion: A-V nicking, copper wire and silver wire changes are usually considered sclerotic arteriolar changes and are commonly attributed to hypertension. Actually the latter two can be shown to occur when the arteriole lies in the stagnant area of a previous vein obstruction. Silver-wire adventitial arteriosclerosis, which has been called arteriolar sclerosis and considered secondary to hypertension, properly should be considered secondary to venous obstruction

and, as such, should be removed from the hypertensive classification. The reason why this arterial change has been misconstrued in the past, and considered due to hypertension is demonstrated by a case of classical secondary arteriosclerosis in a patient with hypertensive retinopathy. A careful search showed an obstructed vein. Aneurysms, new vessels and all the changes seen after vein obstruction were also present. The hypertensive changes, cotton-wool exudates, nerve-fiber edema and flame hemorrhages, were all superficial and tended to hide the evidence of vein obstruction which lay deeper. Leishman has recently demonstrated histologically more venous than arterial pathology in retinas of severely hypertensive patients. A previously unappreciated degree of venous stasis and obstruction occurs in severe hypertensive retinopathy.

What retinal vein obstructions are most responsive to anticoagulant treatment?

Dr. Wise: Most retinal vein thromboses are terminal thrombotic episodes on some other basic vein pathologic process. Only those obstructions of veins draining the macular area have any effect on the eventual visual outcome. Therefore, the first objective in any individual case is to determine the exact site of vein obstruction. Nasal vein obstructions require no treatment. Most of these clear eventually with some depression in the involved field of vision but no impairment of macular function. For the same reason, temporal branch vein obstructions which do not involve macular drainage require no treatment. Occasionally a temporal vein is obstructed at the disc, but the macula is protected by the barrier of a patent vein between it and the obstructed area, draining directly into the central retinal vein. Such a case would require no treatment.

Vein obstructions involving macular drainage which do require treatment may be divided into two groups: (1) branch obstructions and (2) central vein obstructions. My findings are in agreement with those expressed by Klien who has had much more experience than I. Branch obstructions have a poor therapeutic response to anticoagulants. Central retinal vein obstructions which come on gradually and are promptly treated with anticoagulants have a better prognosis than fulminating ones which occur suddenly and those in which onset of treatment is delayed.

It seems important to be on the alert for impending central retinal vein obstructions if we wish more therapeutic success. This is a difficult diagnosis to make but is suggested by dark, dilated, tortuous veins, yellowish discoloration of the fundus and retinal edema exaggerated at the macula. The latter often causes transient blurring of vision or a decreased quality of vision as compared to the fellow eye, when acuities are equal.

Occasionally small hemorrhages about peripheral venous twigs may be seen. If these findings are mild, treatment is not indicated. I have seen them persist for months without actual obstruction. Such patients should, however, be warned to seek advice promptly if there is any sudden reduction in vision and anticoagulants should then be started. When given, anticoagulants should be continued for a minimum of four to six months and probably even longer. They may be given on an ambulatory basis, as an internist would treat a postcoronary occlusion. The effort should be directed toward keeping the obstruction in sufficient abeyance to avoid retinal tissue death while collateral channels develop. Withdrawal of anticoagulants should be gradual.

Discussion. Dr. Linksz: Some years ago, I may have misunderstood this, but it seems to me that I read that Klien said that phlebitic thrombosis should not be treated by anticoagulants. I never knew exactly why this was and I would like to ask Dr. Wise about this and how he establishes what is the cause of the occlusion, just as in that one diabetic case he showed with the central vein occlusion.

Dr. Wise: Diabetic patients have a primary venous disease and are also very prone

to atherosclerosis. It is tempting to think that in this particular patient an atherosclerotic plaque in the artery at the lamina cribrosa led to blockage of retinal inflow, underfilling veins, with subsequent stagnation thrombosis of the central retinal vein.

DR. WAGENER: I am not sure what is meant by phlebitic.

DR. WISE: I believe that Dr. Klien refers to inflammatory obstructions, comparable to thrombophlebitis in the leg. This often occurs during the course of severe illness, generally in a younger group of patients. The relative youth of the patients may be an important factor in their good prognosis, even without therapy. It is probably for this reason that she suggests they should not be treated with anticoagulants. They do well without them.

DR. COSGRIFF: Do you ever see branch occlusion propagate proximally to involve the central retinal vein? You know in peripheral vascular disease people who have a thrombosis in any—say the popliteal artery or the femoral artery—will usually propagate both distally and proximally from the original site. Do you ever see this in the retina?

DR. WISE: I have seen patients develop one branch obstruction, then another, and end with central retinal vein obstruction—each apparently individual episodes. I have never seen what appeared to be true propagation except possibly in Eales' disease where the thrombosis is secondary to the spreading perivenular inflammation.

DR. GIVNER: It is very difficult to evaluate therapy with the anticoagulants in central retinal vein thrombosis. Three cases to show this point:

Case 1. A. G. was seen 15 years ago with a typical picture of thrombosis of the central retinal vein of the right eye. This was before the days of anticoagulants. Nothing special was done. Her vision was 10/200. Today she has 20/30 vision in this eye (she also has an immature cataract to account for this reduction of vision).

Case 2. N. S. aged 70 years, developed a coronary thrombosis. He was put on Dicumarol and his prothrombin time was kept to about 30 or 35 seconds. Four months later while he is still on Dicumarol he developed a complete thrombosis of his only eye and lost his vision.

Case 3. A man who came in also with thrombosis of the retinal vein of one eye, old and finished, had signs suggestive of a prethrombosis of the central retinal vein in his other eye. He had obstruction of vision, absence of pulsation of the vein, and all the signs that Dr. Wise spoke about. He was put on anticoagulant therapy and, after four years, that eye is fine.

Discuss the use of anticoagulant therapy in venous and arterial occlusion of retinal vessels, agents, risks, antidotes, and so forth.

DR. COSGRIFF: Anticoagulant therapy is used in retinal vascular disease because it has given reasonable success in venous thrombosis, pulmonary embolism, coronary occlusion, and cerebral vascular disease. It might be pointed out that there is evidence indicating a hypercoagulable status of the blood in many persons who have acute thrombotic or embolic disease. This hypercoagulable tendency is thought to be one of the important factors in the continuing tendency for propagation of intravascular clot material in patients who have coronary occlusions, pulmonary emboli, venous thrombosis and peripheral arterial diseases. Perhaps this is a factor in retinal vascular disease.

I would also like to point out that there is clinical evidence that propagation of thrombus material, both proximally and distally, as occurs in the veins and arteries of the lower extremity, is a very important mecha nism by which more extensive and important areas of blood vessels are clotted off.

Anticoagulants exert an antithrombotic effect and, when we use them, we actually are using antithrombotic agents, with the intention of bringing about an alteration of the blood-clotting mechanism in such a way that further intravascular clotting is halted but not so excessively as to produce spontaneous

hemorrhage and bleeding. There is also evidence that anticoagulants bring about a more speedy development of collateral circulation. Moreover, there is evidence that the anticoagulants enhance the naturally active fibrinolytic processes of the body, thus bringing about an increase in the rapidity by which some clots are resorbed.

There are two types of anticoagulant agents:

Heparin, given parenterally, has the advantage of being immediately effective and is probably a more potent anticoagulant than is the hypoprothrombinemic group of drugs. However, heparin is more expensive and must be given, as you know, hypodermically. Its administration must be accurately controlled, to prevent the serious effect of excessive hemorrhage, as well as to insure that an adequate antithrombotic effect is being obtained.

The hypoprothrombinemic drugs are those used over a longer period of time, particularly in ambulatory patients. They include such agents as Dicumarol, which is the oldest, and Coumadin, which is one of the newer ones. These all bring about various alterations in the clotting complex. Prothrombin time is needed to estimate the proper dosage of both Dicumarol or Coumadin. These drugs can be given orally and they are less expensive. However, their use must be followed by frequent and reliable prothrombin-time determinations, otherwise they carry a very significant risk of serious hemorrhage.

The risk of anticoagulants and their side-effects are actually only one—hemorrhage, which occurs in about four percent of the patients to whom anticoagulants are administered. The most common complications are hematuria with bleeding from the kidney, nose bleeds, bleeding into the skin, and bleeding into the gastrointestinal tract. The bleeding is actually fairly well controlled by specific antidotes. The antidote for heparin, when one wishes to discontinue heparin therapy, is protamine sulfate. It is of interest that if the venous clotting time should be prolonged to the excessive extent of say two hours, the administration of protamine sulfate, readily available and marketed by one of the drug companies, will return the clotting time to five or 10 minutes within a few minutes of its administration. This allows a large factor of safety in the use of heparin.

A less rapidly effective antidote is available for the hypoprothrombinemic drugs and this is vitamin K_1. Vitamin K_1 is marketed by one of the drug companies as Mephyton. It may be administered by mouth or intravenously. It is rapidly effective when given intravenously. When it becomes necessary to discontinue therapy one can produce a return to a normal prothrombin time within about six to eight hours by using vitamin K_1.

Caution must be used in prescribing anticoagulants. Each patient to be considered for anticoagulant therapy ought to have a substantial review of his medical background. In some instances anticoagulants are really contraindicated and carry more risk of damage than promise of good. Such health problems as peptic ulcer, a recent wound or a healing wound, the presence of liver disease, and other types of intrinsic organic disease, such as renal insufficiency, are situations where there is significant risk in the use of anticoagulants. One real contraindication is the unavailability of reliable laboratory tests. When they are used, the anticoagulants ought to be given in sufficient dosage to bring about an antithrombotic effect, without producing excessive and frequent bleeding complications.

What has been your experience with juvenile diabetics?

DR. GIVNER: Over the past 12 years I have had the opportunity to study cases of juvenile diabetics at Camp Nyda (New York Diabetic Association). In 1,000 eyes, one percent had either pinpoint retinal hemorrhages or small microaneurysmal dilations. In this group, 0.4 percent of the children had cataracts. Dr. Theodore Cohn has studied 600 children, 70 of them over a period of five years. His result shows that 54 percent

show an increase of the beta lipoproteins compared to a group of children with no diabetes who have a 10-percent increase of beta lipoproteins.

What can be done for these poor children, who end up as this child did, completely blind with no light perception? One thing is this. If they have diseased vessels, we must do everything we can to prevent hypoglycemic reactions. For during hypoglycemic reactions, patients with diseased vessels are prone to hemorrhages either in the brain or retina.

We all recognize now that reduction in adrenal cortical function lessens the incidence of diabetic retinopathy. Pituitary necrosis, adrenalectomy and the administration of testosterone (which probably inhibits the pituitary) have all been shown to be of benefit in slowing down and even reversing pathologic retinal conditions, whereas infection and the administration of ACTH have speeded their development in diabetics who have already shown well-developed retinitis proliferans and hemorrhages. Administration of testosterone (25 mg. every seven days intramuscularly) has proven at times to be of much value.

B. Z., a woman, presented herself in June, 1955. The vision of her right eye was 4/200. The left eye showed marked vitreous hemorrhage, retinitis proliferans and vision was 20/100 plus. She had to give up her work. In addition to the routine medication to reduce capillary fragility, testosterone was administered. Up to this time, almost four years, the patient has had over 100 injections of testosterone with no evidence of masculinization. The vitreous is clear. The fundus is free of hemorrhage, there are no exudates, and the vision of the left eye is 20/20− and she is back at work.

What else can be done to cut down adrenal function?

A. B., aged 50 years, had diabetes at the age of 15 years, at which time insulin was started. Today, 35 years later, her blood vessels are normal, and one microaneurysmal dilation is only now beginning to show in the right eye and one punctate exudate in the left eye. On investigating what might be related to her excellent vascular status, it was learned that 11 years after her diabetes started, a denervation of one adrenal was done to see if her diabetes could be cured. Of course, it didn't cure her diabetes but today her ketosteroids are 4.8 mg. (normal, 5.0 to 14 mg.). Could it be that the reduced function of her adrenal, either due to innervational disturbance or incidental adrenal damage, could have had a beneficial effect?

Finally, DuBois found that tetraethyl ammonium chloride in experimental animals protects against the vascular damage caused by the "Shiga toxin." May we not hope that some chemical will be discovered to protect the diabetic patient against the vascular lesions of diabetes? Theodore Cohn believes that such a toxin may be related to the saturated fats in the diet and he suggests that the fats be kept down to 30 to 40 mg. and that unsaturated fatty acids be added to the diet.

Discussion. DR. WAGENER: Recently I have studied another group of diabetics in which 40 cases developed under the age of 12 years and have a duration of nine to 34 years. Fifteen of these 40 cases showed retinopathy; and in 51 percent of these 15, the retinopathy was of a proliferative type. That is a greater percentage of proliferative retinopathy than in the usual diabetic, about 13 percent of whom show retinopathy.

I want to ask Dr. Givner if he has any idea why the percentage of proliferative retinopathies is so much higher in diabetics who started in childhood than in ordinary diabetics whose disease develops at various ages. Is there any particular reason?

DR. GIVNER: I know of no particular reason. It makes quite a difference whether percentages are taken in children who are perfectly normal and would not ordinarily come to see the ophthalmologist or if they are taken in a group of patients who come to the ophthalmologist because they have eye complaints.

One point of interest is that no case of diabetic retinopathy under the age of 10 years has been reported in the literature. There was none in my series either. This means that duration of the disease has a certain influence. Children aged 10 years are probably not old enough to have had the disease long enough to produce the damage that probably would be seen later on. I have also found a high percentage of retinitis proliferans in juvenile diabetics who come to the office with visual complaints. Maybe the young have a greater tendency to gliosis. I don't know. It may be that organic changes in the vessels protect against disease of the retina; that is the only reason I can see why the percentage of proliferation is higher in persons who start as juvenile diabetics.

One other thing—it seems to me I have been told recently that these patients are no longer called juvenile diabetics because of the age at which the disease develops, but rather because of the type of diabetes. Is that true?

DR. RILEY: I wonder about that. I only see cases which develop at an early age; I assumed these were the only cases called juvenile.

DR. COSGRIFF: I go along with this; in other words, it is the time not the type.

CHAIRMAN: I know that diabetics themselves will tell you that they consider any patient a juvenile diabetic who has diabetes under the age of 15 years.

DR. WISE: I'd like to ask a question. Isn't it correct that the juvenile groups show practically no islet tissue and tend to be much more refractory to treatment; a higher dosage of insulin, a really high dosage, has to be used than in older patients, who frequently have normally functioning islet cells and often are treated by some of these newer drugs and can squeeze extra insulin out of the available islet cells.

DR. GIVNER: That is the explanation why children can't be treated with oxinase.

What are the ocular problems in the Riley-Day syndrome of familial dysautonomia?

DR. RILEY: Since many people are not familiar with familial dysautonomia, I ought to mention briefly that it is a peculiar syndrome, of genetic origin apparently, which gives rise to a baby who is a feeding problem from birth, gains poorly, and often has frequent bouts of pneumonia. As he grows older, it becomes apparent that he has several nervous system peculiarities, most pronounced in the autonomic nervous system. For instance, he perspires a great deal, he has an unstable blood pressure, he tends to have postural hypotension, he drools, perhaps he vomits easily and, interestingly enough, he fails to produce tears when he cries and has corneal anesthesia or, at least, hypesthesia. As he grows older, it becomes obvious that he is not very sensitive to pain and the deep tendon reflexes are absent. This is often a familial condition and siblings are also involved. The child is usually of an irrascible disposition so that he presents quite an examination problem.

Ocular problems growing out of this are probably related to the corneal anesthesia or hypesthesia more than to deficiency in tearing. Although these children fail to produce tears when they cry, in general they seem to have normally moist eyes; if they are stimulated by the Schirmer test, for instance, they don't produce excessive tears and they don't respond to lacrimating gases, such as ammonia or onion vapor. The ocular complications usually are seen in the fairly young children, in some cases, under a year and usually in the first few years of life.

The main problem is the development of corneal ulcers, usually probably secondary to abrasions but, in some cases, they appear to be associated with open eyes during sleep. Punctate lesions in the cornea cause corneal opacification. Such ulcers are often very slow to heal. Ophthalmologists who have worked with me on this problem have felt, after failure of healing on conservative therapy, that it is wise to make lid adhesions and keep the eyes closed for a period of at least six months, probably a year or even longer in order to

encourage healing. Some children have developed complete opacification of the cornea and have presented a great deal of difficulty. One thing is important to mention: remember that these children are very poor anesthetic risks. Of 20 or 25 anesthesias administered to such children, complications of some degree have developed in practically all instances and of severe degree in three instances, two of which were complete cardiac arrests saved, I am happy to say, by immediate cardiac massage; in the third, the heart didn't quite stop and started up again spontaneously. In the other cases, there has been difficulty with aspiration, with cardiac irregularities, with blood pressure irregularities; so you should have an anesthetist who knows what he is doing.

One thing, however, that helps is that these children are indifferent to pain and the lid adhesions have been placed in quite a few cases with no more than simple sedation. There is no need to put them under proper anesthesia.

Discussion. DR. GIVNER: How many cases have you seen?

DR. RILEY: We have 110 patients on record of whom about 30 percent have died. Some of them have lived to the age of 27 years.

DR. LINKSZ: In other words, most of them die young? Or many of them?

DR. RILEY: Many of them, rather than most.

What are the evidences of obliterative endarteritis in the ocular fundus? What diseases produce it?

DR. WAGENER: Endarteritis obliterans in the ocular fundi depends entirely in what is meant by endarteritis obliterans. As a matter of fact, as far as I can tell, the term is not used any longer by internists who deal especially with peripheral vascular disease. It appears in ophthalmologic texts, and often it is somewhat difficult to know just what the text means by the term, endarteritis obliterans. If you ask some internists, they say that any inflammation of the artery with closure of the lumen can be called endarteritis obliterans. If that be true, then, of course, we can conceive of such conditions as ischemic optic neuritis or temporal arteritis being manifestations of endarteritis obliterans. Kreibig felt that ischemic optic neuritis may frequently be due to an infarction of the optic nerve, the edema in the disc being a secondary reaction to the localized infarct in the nerve stem.

An arteritis with sufficient closure of the lumen may be called an endarteritis obliterans, or a closed central artery of the retina in temporal arteritis. The peripapillary arteriovenous anastomoses of pulseless disease may be indicative of endarteritis obliterans. Another form is the type of arterial closure that follows the lodgment of an embolus, a septic embolus, or a nonseptic embolus occurring in lupus erythematosus with Libman-Sacks disease, nonbacterial vegetative endocarditis.

On the other hand, in most ophthalmic texts, for instance in Duke-Elder and in Fuchs' and Samuels' *Atlas*, what is called endarteritis obliterans, following the terminology used by Coats, is really atherosclerosis. Then, of course, if that interpretation of the term is accepted, any fundus change that goes along with atherosclerosis, for instance, closure of the central artery of the retina, can be included.

A case of ischemic infarction of the retina, with fundus evidence of atherosclerosis (perhaps an illustration of the propagation of a thrombus that Dr. Cosgriff was talking about) started, in my opinion, as an atheroma in the central artery and a superimposed thrombus propagated down into the lower branch. The rest of the retina cleared up, but the inferior temporal sector remained ischemic and subsequently became atrophic. Of course, there can be closure of the central vein of the retina secondary to atherosclerosis of the central artery. I used to consider certain white patches on arterioles to be evidence of periarteritis. I believe now that they are manifestations of the fibrotic stage of

atherosclerosis, as explained by Friedenwald in his *Atlas*.

A similar but very much more extensive arteriolar lesion resembles those which Dr. Wise thinks develop subsequent to a venous obstruction. However, this patient had never had, up to the time this photograph was taken, any visual impairment of any sort and we could find no evidence of venous disease other than the relatively dilated vein. That blood is visible in the arteriole distal to the plaque indicates that the arteriole is not occluded.

An atherosclerotic closure of the superior temporal arteriole with residual secondary occlusion of the superior temporal vein would be called arteriosclerotic retinopathy. However, the whitening of the superior temporal arteriole is quite possibly secondary to the retinal changes resulting from the occlusion of the vein. I agree with Dr. Wise on this point. Personally, I do not believe that there is a true arteriosclerotic or arteriolosclerotic retinopathy. I think that, in all such cases, if you examine them closely enough or at the right time, you will find evidences of venous obstruction, either of a large vein or of a small venule. This type of small punctate exudate and small punctate hemorrhages do not result from purely arterial or arteriolar lesions in the retina but always are evidence of previous venous obstruction.

It is claimed that nontraumatic macular holes are due to endarteritis obliterans, now spoken of as arteriosclerosis obliterans, in the small arterial vessels leading toward the macula.

Of course, evidence of arteriosclerosis obliterans cannot be seen in the choroid. There are two types ophthalmoscopically visible in the vessels. I have tended to call the varying degree of color change sometimes seen in the choroidal vessels senile choroidal arteriosclerosis. It may be associated with atherosclerosis elsewhere in the body and may underlie some of the choroidal degenerations. In cases of hypertension, single, individual white choroidal vessels may be seen. These also may be due to atherosclerosis but many of them, I think, are due to medial hypertrophy and closure of the lumen, as seen in microscopic sections of hypertensive retinopathy.

At the present time, men interested in the general peripheral vascular system say that endarteritis obliterans is now spoken of as arteriosclerosis obliterans, and that about 90 percent of these cases are atheromatous. On the other hand, what was originally called by general pathologists endarteritis obliterans is now called thromboangiitis obliterans, Buerger's disease. Is there anything in the eye to indicate Buerger's disease? This is a very difficult question to answer. In the first place, we do not see too many eyes of patients with Buerger's disease. These patients come in to the peripheral vascular man, usually with complaints such as intermittent claudication, and, unless they have a specific eye complaint, they are not seen by the eye man except by accident.

Several years ago, I saw a case of what would be called Eales' disease, recurrent hemorrhages into the retina and vitreous, of unknown cause. That same patient came back about four years later with typical Buerger's disease of the extremities. He still had active lesions of periphlebitis with hemorrhages in the retina, but he had not had any large hemorrhages into the vitreous at the time he was back for the second visit. Some years ago I saw a few cases of retinal venous occlusion in patients with Buerger's disease but I have not seen any in recent years. I have started to review histories of patients with Buerger's disease. I have not yet been over the whole series because it is difficult to find records of eye examinations. In about 100 histories 50 had no record of eye examination. Of 50 who had the maority had some hypertension along with the Buerger's disease, and showed ordinary hypertensive changes in the retina. There was one patient, however, who, when first seen, had only a few hemorrhages in the retina, with some suggestion of venous stasis. I

thought he might be a diabetic but he proved not to have diabetes; he had had Buerger's disease for 15 years. He came back about two years later with what was thought to be cerebral Buerger's disease. There was generalized cerebrovascular disease with multiple infarcts, and for that reason he had his fundus examined. He showed a recent occlusion of one arteriole, with ischemic edema of the retina, recent occlusion of one vein with large hemorrhages in the retina, as well as evidences of periphlebitis and periarteritis in other vessels of the retina.

Another patient some four years before, at a rather young age, about 30-some-odd, had had complete occlusion of the central retinal artery of one eye, with resultant blindness. At the time that I saw him, he had optic atrophy in the blind eye and, in the other eye, phlebitis of the superior temporal vein with hemorrhages in the retina. There was also a possible thromboangiitis obliterans in one arm. In the group I reviewed there were two cases in which the presumptive diagnosis of cerebral Buerger's disease was made. In one of these cases there was bilateral papilledema and in the other, homonymous hemianopsia. The only other case with positive eye findings, aside from retinal arteriolosclerotic lesions, was that of a patient who had a toxic retinopathy; however at necropsy that patient was proved to have dermatomyositis. The probability is that the retinopathy was due to the dermatomyositis rather than the thromboangiitis obliterans.

On the basis of these findings, it seems reasonable to conclude that it is possible for thromboangiitis obliterans to involve the retinal vessels. Possible evidence in the eye of Buerger's disease is a retinal lesion in which, as is the case in the peripheral vessels, there is at the same time arterial and venous involvement not in companion vessels. You can see in this slide that the occluded arteriole is not in the same area of the retina as the venous lesion; in the fundus photographs Dr. Wise showed the arteriolar and the venous lesions were in the same quadrant of the retina. I think that the main reason for considering such vascular lesions as probably thromboangiitis obliterans of the retinal vessels is that, in Buerger's disease of the peripheral vessels, it is rather characteristic to see this combination of occlusive arterial disease associated with thrombophlebitis. In the case shown, the occluded arteriole in the inferior temporal quadrant and the venous lesion in the superior temporal quadrant of the same retina would have to be considered as separate manifestations of the same vascular disease. This was one of the first cases in which we thought that the diagnosis of thromboangiitis obliterans of the retinal vessels was probably justified.

What is the present concept of retinitis proliferans? Does this have application to any systemic disease?

Dr. Wise: Retinitis proliferans is the growth of new blood vessels and their associated fibrous scaffolding into the vitreous. It has long been considered due to the organization of pre-existing vitreous hemorrhage.

Ballantyne divided retinitis proliferans into two types: reté and fibrotic. In the reté form the proliferation is predominately vascular. Delicate new vessels of capillary structure but wider lumen, with their associated, scarcely visible, fibrous scaffolding, grow on the inner retinal surface. In this form where vascular connections have been demonstrated histologically, these new vessels arise from obstructed capillaries or small veins. In the second type, fibrous proliferans, fibrosis predominates and vessels are larger but fewer in number. These vessels also have been shown histologically to arise from obstructed retinal veins.

Ballantyne (in diabetes) and Ashton (in retrolental fibroplasia) have demonstrated microscopically that reté proliferans may develop without pre-existing vitreous hemorrhage. Anyone sufficiently patient to watch the development of diabetic retinopathy with the retinal slitlamp, frequently repeated examinations over a prolonged period being

necessary, see both reté and fibrotic types develop and extend into a clear vitreous without pre-existing hemorrhage. Now, if not only reté but also fibrotic proliferans develop prior to the onset of vitreous hemorrhage, what then is the stimulus for the new vessel growth?

The unicellular organism has no vascular system. It carries out physiologic exchange with its environment through the cell wall. As one moves up the phylogenetic scale the organism becomes more complex. The inner cells become unable to carry out this exchange with their environment. At this stage the vascular system develops. Thus, vessels arise in response to inner cellular need.

The changing vascular patterns seen in the developing embryo are primarily functional adaptations to the ever-changing cellular needs. At any given moment the pattern represents a physiologically appropriate system for the tissues under prevailing conditions. When an individual organ is growing rapidly, a capillary plexus grows out from regional blood vessels to meet the local cellular requirements. Those capillaries carrying blood to the cells later become arterioles; those carrying blood from the cells, venules. This differentiation into artery or vein is a latter development and seems to be dependent on the direction and rate of blood flow and the intraluminal pressure. The arterialization of a vein wall in cases of arteriovenous fistula demonstrates that this adaptive ability does not disappear from mature vessels. As cellular requirements are adequately fulfilled, certain vessels may atrophy or disappear. The ligamentum teres may be considered an example of this.

Thus, both phylogenetically and embryologically blood vessels develop in response to tissue or cellular need. What then occurs in the adult retina with its mature vascular system to make new vessels suddenly develop?

The trigger mechanism in retinitis proliferans appears to be obstruction of capillaries or veins, irrespective of the etiology of the obstruction. With either capillary or vein obstruction a state of circulatory impedence follows, a relative hypoxia in the stagnant region develops, and tissue cells in such areas are adversely affected but do not die. Arterial obstructions, on the other hand, in such an end-artery system, lead to infarction and cell death. In the former, new blood vessels develop in response to the hypoxic retinal cells; in the latter, cell death eliminates the need of new vessels and they do not appear.

Fibrotic proliferans can be seen to develop clinically in two ways, neither of which is dependent on antecedent vitreous hemorrhage. Reté proliferans tends to become less vascular and more fibrotic with the passage of time. The early and late stages of retrolental fibroplasia are examples of reté and fibrotic proliferans respectively. With obstruction of large retinal veins or of the central vein, the proliferans is usually fibrotic from the beginning.

Retinitis proliferans is a purposeful attempt to restore adequate circulation to meet the needs of hypoxic retinal cells.

Before applying this concept to any systemic disease process, let us review its essential features. First, there is capillary or vein blockage leading to stagnation and hypoxia of retinal cells. Thus a cellular need for a better vascular supply is established. Next, in response to this cellular need, new blood vessels grow out to restore the circulation. In early stages, new blood vessels tend to predominate. Later, as the more direct channels are favored, superfluous vessels atrophy and contracting fibrosis remains. And finally, as is readily visible with the slit-lamp, a fringe of avascular fibrous tissue always precedes growing new vessels. Thus fibrosis precedes new vessel formation and remains after many such vessels have disappeared.

Let us apply these principles to the development of cardiac cirrhosis. First, we have congestive failure and chronic passive congestion of the liver with its dilated, engorged venous sinusoids and central lobular veins. With this impedance of venous circu-

lation, regional cells around the central lobular vein are most adversely affected. After a period of time these cells die, producing a central necrosis. The more peripheral cells in the liver lobule are adversely affected but do not become necrotic. They remain viable but usually exhibit fatty degeneration. In response to the cellular need in each liver lobule due to liver cell hypoxia, fibrosis and probably—I say probably because it has been very seldom demonstrated—an attempt at new blood vessel formation commences and grows out in tenuous shreds through the sinusoidal spaces toward the more adequately oxygenated arterial periphery of the liver lobule.

If the cardiac failure is progressive and longstanding, the final picture is one of marked central lobular fibrosis with a thickened fibrous central lobular vein and considerable endothelial proliferation. Strands of fibrosis peter out as they extend through the sinusoidal spaces toward each lobule periphery. This is quite comparable in many respects to the final picture of severe retrolental fibroplasia. If, however, the cardiac failure improves, more central liver cells recover with improved oxygenation and no longer stimulate fibrosis and new vessel formation. The fibrous tissue regresses, the sinusoidal bands become narrower, fibroblasts disappear, and any attempt at new vessel formation ceases. This is quite comparable to the regression and even disappearance of both new vessels and fibrous tissue as has been reported in spontaneously arrested cases of Eales' disease.

Time does not permit us to apply these principles to other forms of cirrhosis, or to the patchy fibrosis of lung, heart, spleen, kidney or cirrhosis reported in sickle-cell disease where red blood cells become rigid at lower oxygen tensions, thereby mechanically blocking capillaries and small venules. This basic mechanism may well be applicable in some cases of interstitial lung fibrosis, in the pathologic process of phlebopathic leg ulcers, and even in simple wound healing. It has long been overlooked in the eye where the changes can be readily visualized even while they are taking place. How much more difficult it will be to prove this a proper concept in these opaque tissues where we have great difficulty seeing capillaries and venules even in the normal histology.

Alan H. Barnert,
Recording Secretary.

Ophthalmic Miniature

Three nevi, two in the skin of the right upper, the other in that of the left lower lid, were treated by drawing floss silk, steeped in perchloride of iron, through them. The following inflammation was slight; the threads were removed the third day. One of the nevi was cured within a month, one in three weeks, and a small remaining portion of the third was removed by ligature.

Dr. Bader,
Royal London Ophthalmic Hospital Reports, **2**:113, 1859-1860.

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THE PRESENT STATUS OF DESENSITIZATION THERAPY IN OCULAR TUBERCULOSIS

In an editorial in THE JOURNAL in 1941, the story of tuberculin—its discovery by Koch, the high hopes that it might be the dreamed-of remedy for tuberculosis, its use as a therapeutic agent under the old perifocal concept of its action, the discouraging and often disastrous results observed, the gradual abandonment of such therapy, the resurgence of its use under the desensitization concept—were briefly summarized. Certainly from 1929 to 1948, desensitization with tuberculin was the most useful weapon in the ophthalmologist's armamentarium in the battle against the ravages of ocular tuberculosis.

With the advent of specific antibacterial therapy against the M. tuberculosis with streptomycin, paraaminosalicylic acid, and isoniazid, desensitization with tuberculin has fallen into comparative disuse. The immediate question is whether tuberculin still has a place in the treatment of ocular tuberculosis, or whether in the light of new developments, it should be abandoned and forgotten.

In the consideration of this question, it must be remembered that no toxic component has ever been isolated from the tubercle bacillus and it does not produce an exotoxin. After infection of previously normal tissues there is at first a minor inflammatory reaction. Thereafter an increased resistance to the infection develops, and at the same time the tissues become hypersensitive to the specific protein of the bacillus. Once such sensitization has occurred, there is a reaction between the sensitized tissue and the specific antigen—the protein fractions of the bacilli. This reaction is accompanied by acute inflammation, and if the bacilli are sufficiently numerous, by necrosis and caseation. This is the destructive phase of the tuberculous lesion.

The essential principle underlying tuberculin desensitization is the removal of the dangerous tissue hypersensitivity. It must be remembered that resistance to infection and hypersensitivity are separate and distinct immunologic phenomena, and that the tissue hypersensitivity can be abolished and removed while the resistance to infection remains undisturbed. Desensitization is accomplished by the parenteral injection of antigen in quantities so small that focal reactions around the lesions are avoided. As the tissue sensitivity is thus depressed, the antigen is administered in gradually increasing amounts, taking care to keep the dose below that which will cause any local or systemic reaction. By this means hypersensitivity can be greatly depressed, or even so completely abolished that contact of the tissues with the living bacilli or their protein products is not accompanied by a reaction sufficient to produce secondary inflammatory lesions in the desensitized tissues.

If antibacillary therapy in tuberculosis were 100-percent effective, and all the bacilli were destroyed by the bactericidal agents and no living bacilli remained in the encapsulated lesions, obviously the long drawn-out process of desensitization with tuberculin would be unnecessary and unjustified. Unfortunately, complete destruction of all bacilli is not always the case.

When experimental animals with a secondary ocular tuberculosis are treated with antibacterial therapy, clinical improvement is usually observed anywhere from the seventh to the fourteenth day of treatment, and after six weeks of treatment all clinical evidences of activity have usually disappeared, leaving scarred but inactive eyes. However, if the antibacterial therapy is now discontinued and the animals kept under observation for a six-month period and then killed, there is a full 20-percent failure rate, including clinical recurrences, histologic evidences of activity, or positive cultures of the macerated uveal tracts. In these animals, despite the apparent clinical recovery, living bacilli still remained in the encapsulated lesions. In humans, who have what appears to be undoubted ocular tuberculosis, much the same phenomena are noted. Clinical improvement in the active uveitis or kerato-iritis is usually noted from the seventh to the 14th day, and the eyes usually appear quiet to clinical examination after six weeks of the antibacterial therapy. In fact, if significant clinical improvement is not noted by the 21st day of treatment, the etiologic diagnosis may well be questioned and the case re-evaluated. However, in patients receiving only six weeks' treatment, there is again a 20-percent failure rate, manifested by later recrudescences or exacerbations of the disease.

Mitchell, in an editorial in THE AMERICAN JOURNAL OF OPHTHALMOLOGY in 1955, noted this failure rate, and using the analogy of urogenital tuberculosis where the urine may be cultured for viable bacilli, suggested

that prolonged antibacterial therapy in patients with ocular tuberculosis, after all clinical evidence of activity has disappeared, might radically reduce this high percentage of failures. Accordingly, it is now the usual custom to continue the antibacterial therapy in successfully treated cases for a minimum of six months, or more properly even for a full year.

It is undeniable that this prolonged antibacterial therapy has materially reduced the failure rate in human ocular tuberculosis. However, while there are no exact figures, it is also true that even after such protracted treatment occasional relapses or recrudescences of the ocular inflammation do occur. There is no guarantee that even a full year's antibacterial treatment will kill all the bacilli in the encapsulated lesions within the eye, and there is no surety that there may not be a later depression in the host's resistance. With such a depression the latent organisms may again propagate in the tissue. If this multiplication of the bacilli takes place in sensitized tissue, it will be accompanied by acute inflammation, necrosis and caseation.

Thus, in essence, desensitization therapy as formerly employed without concomitant antibacterial therapy was not a curative procedure, but was designed to remove the tissue sensitivity which is the factor responsible for the destructive phases of the tuberculous lesion. If employed in conjunction with the modern antibacterial therapy, it must be regarded as a prophylactic measure, designed to abolish this same dangerous tissue reaction, and to lessen the dangers of acute inflammation in the event the antibacterial therapy should prove to be ineffective in destroying all the latent bacilli, and there should ensue a later recrudescence of the ocular disease.

Since recurrences of ocular tuberculosis do occasionally occur even after prolonged antibacterial therapy, and since tuberculin desensitization, properly administered, is a harmless form of treatment and may be given concurrently with the antibacterial therapy, it would appear unwise to consign this proven and time-honored treatment to the limbo of obsolete therapeutic procedures. It may well be employed in all cases of ocular tuberculosis as an adjuvant to antibacterial therapy. In cases where organic damage has already been done to the eye and the margin of safety has been thereby proportionately reduced, and where a minimal lesion would result in an irreparable loss of function, it is a thoroughly justified and definitely indicated procedure.

Alan C. Woods.

INDIRECT OPHTHALMOSCOPY

The recent revival of the interest in indirect ophthalmoscopy is noteworthy. This is in part the result of the development of the binocular electric ophthalmoscope and its use in the study of the fundi of cases of retinal detachment. Indirect ophthalmoscopy, including binocular indirect ophthalmoscopy has been known for many years. Up to the advent of the first practical battery handle electric ophthalmoscope in 1913, the use of the reflecting ophthalmoscope for direct and indirect ophthalmoscopy was routine in every eye case examined. The light source for these reflecting ophthalmoscopes came from a candle; later an oil lamp; then a gas light and finally an electric globe.

The ophthalmoscope was invented by Babbage, the inventor of the calculating machine, working with an English medical student, in 1848. Its use, however, was neglected and the instrument was forgotten.

The discovery of the ophthalmoscope was revealed by Hermann von Helmholtz in a paper read before the Berlin Physical Society the night of December 6, 1850. The paper had the unrevealing title "About the luminosity of the eyes." The contribution, however, was of utmost importance for it described a method of examining the interior of the eye. The circumstances leading to the discovery of the ophthalmoscope are not without interest.

In 1849, Helmholtz was appointed to the chair of physiology and general pathology in Königsburg. In this position he gave much of his time to teaching physiology and some of the lectures were devoted to the sense organs. The eye was one of the subjects that was considered in detail. Helmholtz and the other physiologists of the time were interested in trying to determine why under usual conditions, the pupil of the eye appears black and light seems not to be emitted, while under certain unusual conditions light is emitted and the interior of the eye assumes a reddish appearance. In 1847 Brücke in Vienna had been able to demonstrate that the luminosity of the eye was the result of light being reflected from the retina.

It was in an effort to demonstrate this fact to his students that Helmholtz did the work that led to the discovery of the ophthalmoscope. This accounts for the title of the original presentation. Helmholtz, however, soon recognized the importance of this instrument in ophthalmology as evidenced by a letter to his father written in December of 1850 in which he states: "As a consequence of my researches on the physiology of the sense organs, I have constructed a device which should prove to be extremely useful in the science of ophthalmology. This invention is so simple that it is ridiculous to me now that other people as well as myself could have been so stupid as not to discover it before . . ." When the realization of its importance became apparent to Helmholtz, he, in 1851, published a monograph entitled "Description of an eye mirror for examination of the retina in the living eye" (ophthalmoscope). Following this description there was developed a general interest in ophthalmoscopy. This resulted in the invention of many ophthalmoscopes of different design.

In 1852, Ruete invented the indirect method of ophthalmoscopy. This was quickly adopted to be used in conjunction with direct ophthalmoscopy and most instruments had a double set of mirrors so that one could change quickly from one type to the other. With the advent of the electric ophthalmoscope indirect ophthalmoscopy fell into disfavor especially after Crampton in 1913 presented the battery handle type of illumination.

The first attempt to develop a binocular ophthalmoscope was the instrument invented by Girard-Teulon in Paris in 1861. The instrument, however, was never really successful. Binocular direct ophthalmoscopy was made practical with the development of the Zeiss binocular demonstrating ophthalmoscope. The instrument was good but had so many adjustments that if one of these were out of alignment it took some time to get the instrument in good focus again. The instrument developed by Bausch and Lomb also had a vogue for a time. At present, these have been superseded by the Hruby lens and slit-lamp for binocular direct ophthalmoscopy.

With the development of more illumination in ophthalmoscopes using the electric current, such as the giant scope, indirect ophthalmoscopy with the luminous ophthalmoscope became easily available and most of the ophthalmologists who had been trained with the reflecting ophthalmoscope used the method regularly as part of their examination. However, the use of indirect ophthalmoscopy by the generation trained in the use of the electric ophthalmoscope received its impetus from the work in retinal detachment. This has been further stimulated by the binocular luminous indirect ophthalmoscope developed by American Optical Company working with Schepens. This has resulted in a revival of indirect ophthalmoscopy with a tendency on the part of some of the younger men to discard direct ophthalmoscopy.

With indirect ophthalmoscopy the image is inverted and the picture is smaller than in the direct method. The size of the inverted image varies with three factors: the power of the lens used, the state of accommodation of the observed eye, and the static refraction of the same. There are certain advantages in using indirect ophthalmoscopy. In

high myopia, the fundus can be seen easily and clearly. Where the media are cloudy one can often see fundus changes that are not visible with direct ophthalmoscopy. It is also very useful in the examination of children when ophthalmoscopy is done without an anesthetic. With a little patience, even in a rather unco-operative child, it is usually possible to get a good view of the fundus. It is particularly valuable in the examination of the periphery of the fundus as in retinal detachment work. With the smaller image but the larger field it makes it easier to get an over-all picture of the fundus. And finally there are times when it is an advantage to put greater distance between the patient and the observer.

There are, however, certain disadvantages in the use of indirect ophthalmoscopy. While the field is larger the individual objects in that field are smaller and while sharply defined the finer details are seen less perfectly due to the lower magnifying power. The differentiation between objects of similar appearance is less perfect. The difference between veins and arteries is not so marked as with the direct method, and it may be almost impossible to distinguish from each other the finer twigs of each system. The light streak so noticeable in the upright image is frequently wanting. The macular region, especially if the pupil is not dilated, presents unusual difficulties in its examination. If the patient is told to look directly into the ophthalmoscope, this illumination of the macula causes a contraction of the pupil and this causes confusing reflexes.

From this it is apparent that indirect ophthalmoscopy, even with the luminous binocular ophthalmoscope, is not a substitute for direct ophthalmoscopy. It does, however, form a valuable adjunct to direct ophthalmoscopy and at times makes it possible to provide information to the internist, the neurosurgeon and the pediatrician which they are unable to obtain, even though most of them use the direct ophthalmoscope very well.

The American Board of Ophthalmology examinations the last few years have brought out the fact that ophthalmoscopy is one of the poorer subjects as far as the grades are concerned. It is interesting that many of these candidates who had difficulty in the examination could diagnose the condition when it was shown to them on a fundus photograph but failed to find a similar lesion when asked to examine the patient. Thus it would seem well to stress the importance of teaching ophthalmoscopy, but both direct and indirect, by having the resident study patients rather than photographs.

A plea is made to our training centers that the residents be properly instructed in the use of indirect ophthalmoscopy with the electric ophthalmoscope, either monocular or binocular and that its importance as an adjunct to direct ophthalmoscopy be emphasized.

Frederick C. Cordes.

CORRESPONDENCE

Retrolental fibroplasia

Editor,
American Journal of Ophthalmology:

With the marked drop of incidence rate of retrolental fibroplasia, interest in this disorder has virtually disappeared. Except for the reports of certain state agencies for the blind, almost no retrolental fibroplasia data are being published and probably very few are being collected. Our retrolental fibroplasia research group at the Massachusetts Eye and Ear Infirmary, however, has been working since 1954 on a five-year follow-up study of mild and severe retrolental fibroplasia at the Boston Lying-in Hospital and has, as part of that study, followed the changes in incidence during the period when restrictions on the use of supplementary oxygen were being introduced into premature-infant care.

Table 1 presents the incidence rates of mild and severe retrolental fibroplasia for 930 infants of birth weight four pounds or

less, born at the Boston Lying-in Hospital during 21 years, from the time of the appearance of the hospital's first known case of retrolental fibroplasia in 1938, until the end of 1958.

The figures for the severe, irreversible form of the disease are given for the entire 21 years. During this period, 105 (11 percent) of 930 infants developed severe retrolental fibroplasia. In the 10 years from 1949 through 1958, 123 (23 percent) of 537 children developed mild retrolental fibroplasia. (Data for the mild, reversible form exist only since 1949 when, following the lead of Owens in Baltimore, we started regular biweekly ophthalmoscopic examinations of all infants of birth weight four pounds or less, and witnessed either the full development of the severe form, or the halt and regression of the mild form. Before 1949, retrolental fibroplasia was diagnosed only when the resulting blindness became evident; it was known, therefore, only in its severe, full-blown form.)

It can be seen from the table that the incidence rates of both forms of retrolental fibroplasia have indeed dropped. But it can also be seen that occasional cases still occur. From the records of the Boston Lying-in Hospital we know that each year fewer babies have received supplementary oxygen, and for shorter periods. In 1954, 88 percent of the infants of birth weight four pounds or less were given supplementary oxygen; by 1958, only 48 percent received oxygen therapy. In 1954, the average number of hours of oxygen therapy was 92 hours per infant; by 1958, it had been reduced to 15 hours per infant. It is not possible to know whether the extreme of reduction of oxygen therapy has been reached, but it is probable that there will always be some infants who require supplementary oxygen for survival, and from among these will come, perhaps, some of those cases of retrolental fibroplasia which continue to appear. In addition, we have found, during the past few years, several cases of retrolental fibroplasia in infants who, so far as we have been able to learn from a thorough search of the hospital rec-

TABLE 1

INCIDENCE RATES FOR MILD AND SEVERE RETROLENTAL FIBROPLASIA IN A GROUP OF 930 INFANTS OF BIRTH WEIGHT FOUR POUNDS OR LESS BORN AT THE BOSTON LYING-IN HOSPITAL, 1938–1958

Year	Number of infants	Incidence of Mild Retrolental Fibroplasia		Incidence of Severe Retrolental Fibroplasia	
				Percent	(Infants)
1938	22			18	(4)
1939	25			4	(1)
1940	32			3	(1)
1941	34			0	(0)
1942	37			11	(4)
1943	35			23	(8)
1944	34			26	(9)
1945	37			32	(12)
1946	55			14	(8)
1947	40			15	(6)
1948	42			26	(11)
		Percent	(Infants)		
1949	65	23	(15)	14	(9)
1950	64	39	(25)	8	(5)
1951	66	27	(18)	15	(10)
1952	50	36	(18)	12	(6)
1953	37	38	(14)	3	(1)
1954	50	28	(14)	8	(4)
1955	51	12	(6)	2	(1)
1956	60	12	(7)	7	(4)
1957	46	4	(2)	2	(1)
1958	48	8	(4)	0	(0)
TOTAL	930		123 infants		105 infants

ords, received no supplementary oxygen. Five such infants developed mild retrolental fibroplasia and one, severe retrolental fibroplasia. In addition, two infants showed mild retrolental fibroplasia after only 11 hours in oxygen; one severe case developed in an infant who received supplementary oxygen for only seven hours. These occasional cases in infants who received no oxygen therapy suggest that some other causative mechanism—rare, to be sure—exists.

The clinical problem of retrolental fibroplasia has in large part been solved, by the restriction of oxygen therapy, but an awareness that *some* retrolental fibroplasia still occurs should, I think, be kept in our minds.

(Signed) Leona Zacharias, Ph.D.,
Boston, Massachusetts.

CARBONIC ANHYDRASE

Editor,
American Journal of Ophthalmology:

May I ask if there is any means of communication whereby I can record my disagreement with the conclusions of a recent paper in THE AMERICAN JOURNAL OF OPHTHALMOLOGY? I refer to the work of Green and Sawyer in volume 48, no. 1, part II, page 71. The problem is briefly as follows:

Green and Sawyer conclude that the action of Diamox in the rabbit eye has nothing to do with carbonic anhydrase or its inhibition. You will agree that if this is true it is a matter of considerable importance. However, none of the data in this paper point in this direction, although it is given a prominent place in the discussion and conclusion. Evidence for this alleged point comes from their reference to their own earlier study in the Archives of Ophthalmology, Volume 52, Page 758, that is reference 7, which is frequently cited in the present paper.

Concerning this reference 7, Green and Sawyer state that the rate of transfer of bicarbonate ion into the posterior chamber and the rate of flow of aqueous humor are reduced within five minutes after injection of Diamox, at which time the carbonic anhydrase activity of the anterior uvea was not appreciably affected. The latter phrase is the point at issue, for in their earlier paper, Green, et al. have no data on carbonic anhydrase activity five minutes after injection of Diamox! Their first point comes at 15 minutes, at which time 75 percent of the enzyme is inhibited.

In another study which Green and Sawyer do not quote, Ballintine and Maren (Am. J. Ophth., volume 40, page 148) showed that 10 mg./kg. of Diamox (one-third the dose that Green and Sawyer used) abolished all demonstrable enzyme activity in the ciliary processes 20 minutes after injection, and that concentration of the drug in the anterior uvea was about 100 times greater than that needed for complete inhibition for the enzyme.

In summary then, Green and Sawyer in the present paper refer to material from their earlier work, which simply does not exist in print. In fact, they distort their earlier findings and conclusions which suggested that most of the enzyme activity was abolished. Secondly, they ignore other work which shows complete inhibition of the enzyme in the same species, using the same drug.

It is somewhat surprising to me that these matters were not caught by your reviewer, since a glance at either the earlier paper by Green, et al., or by Ballintine and Maren would have shown immediately that a considerable burden of proof lies with anyone claiming that carbonic anhydrase is not involved in the action of Diamox on the eye. I certainly agree that much more remains to be learned in this field and that Green and his colleagues have posed some interesting problems. For example, is carbonic anhydrase involved in the formation of bicarbonate ion or merely its transfer from plasma to aqueous humor? But nothing is gained by the unjustified presumption that the drug does not affect carbonic anhydrase. Because of the importance of this problem

in electrolyte physiology and particularly in ophthalmology, I strongly urge that Green's comments should not go unchallenged.

(Signed) Thomas H. Maren, M.D.,
Gainesville, Florida.

DR. GREEN'S REPLY

Editor,
American Journal of Ophthalmology:

Dr. Maren has claimed that "a considerable burden of proof lies with anyone claiming that carbonic anhydrase is not involved in the action of Diamox on the eye." By the same token, I am sure Dr. Maren will agree that the validity of a theory explaining the mode of action of a drug upon physiologic processes is determined by the extent to which the experimental observations are compatible with the theory. To be sure it is equally incumbent upon those who claim that carbonic anhydrase is the enzyme chiefly responsible for the responses elicited by Diamox on the eye to support their contention with convincing and conclusive evidence. In my opinion the following discussion represents the situation with regard to the dynamics of aqueous humor formation and the effect of Diamox thereon.

It is axiomatic that the basic, although not necessarily the only, requirements for the participation of an enzyme as a primary determinant of physiologic processes are: (1) the reaction mediated by the enzyme is fundamentally related to the physiologic processes; (2) inhibition of the enzyme activity results in a disruption or suppression of the physiologic processes, dependent upon the enzyme activity, with either an accumulation of the substrate of the reaction or a disappearance or decrease in the concentration of the product of the reaction; (3) the existence of a good correlation between the rate of the physiologic processes and the rate of the enzymatic reaction.

The physiologic processes under consideration are the formation or flow of aqueous humor in the rabbit eye and the accompanying maintenance of normal intraocular pressure. The enzymatic reaction presumed to be directly involved in these physiologic processes is the formation of bicarbonate ion in the aqueous humor of the posterior chamber resulting from the hydration of CO_2 to H_2CO_3. The latter reaction has been shown, in in vitro experiments, to be catalyzed by the enzyme carbonic anhydrase.

The history of the development of the theory that carbonic anhydrase in the ciliary processes is chiefly responsible for the elaboration of the bicarbonate ion in the aqueous humor from CO_2 in the plasma, and, therefore, immediately involved in the formation of aqueous humor, has been discussed by Green and Sawyer (Papers No. VI and VII of a series, Am. J. Ophth. **47**: Jan., pt. II, 530, 1959; ibid. **48**: July pt. II, 71, 1959), and need not be repeated here. However, in connection with the present problem raised by Dr. Maren, it is pertinent to recall that the evidence in support of the carbonic anhydrase theory of formation of aqueous humor is: (1) the intravenous injection of Diamox, a potent inhibitor of carbonic anhydrase, was shown by Green and coworkers (A.M.A. Arch. Ophth. **52**: 758, 1954) to inhibit the enzyme in the anterior uvea of the rabbit eye; (2) intravenous Diamox produced a lowering of the bicarbonate ion concentration in the aqueous humors; (3) intravenous Diamox reduced the rate of flow of aqueous humor and the intraocular pressure. The indicated apparent correlation between the pharmacologic responses and the chemical changes elicited by the drug suggested a pivotal role for the enzyme.

The first experimental evidence not compatible with the carbonic anhydrase theory of the formation of aqueous humor, the maintenance of intraocular pressure and the elaboration of bicarbonate ion in the aqueous humor was provided by Green and coworkers (Am. J. Ophth. **40**: Nov., pt. II, 137, 1955) who found that the subconjunctival injection of Diamox completely inhibited the carbonic anhydrase activity of the an-

terior uvea of the rabbit eye, without any apparent effect either upon the bicarbonate ion concentration of the aqueous humor, or upon the rate of flow of that fluid and the intraocular pressure. This evidence seems to have been ignored or conveniently dismissed by Dr. Maren as not being important in the evaluation of the validity of the carbonic anhydrase theory.

In a later study of the kinetics of the dynamics of Diamox action upon the rabbit eye, Green and coworkers (Am. J. Ophth., **44**: Nov., pt. II, 388, 1957) pointed out a lack of correlation between the biochemical parameters of the aqueous humor and the pharmacologic responses that could not be reconciled with the carbonic anhydrase theory of the action of Diamox upon the rabbit eye.

It was apparent to me, therefore, that the case for the carbonic anhydrase theory of the formation of aqueous humor and of the associated postulated source of the bicarbonate ion, as well as of the presumed mode of action of Diamox, was not only inconclusive but also not compatible with all of the experimental facts. Consequently, the experiments with C^{14} labeled $NaHCO_3$ were carried out by Green and Sawyer in order to subject the postulated theory to a more critical and fundamental evaluation (Papers No. VI and VII of series).

Unfortunately, Dr. Maren has either overlooked or did not consider important the results of Green and Sawyer (Paper No. VI) using C^{14} labeled $NaHCO_3$ with normal, untreated rabbits. In this regard it is particularly pertinent to remind Dr. Maren that on the basis of both theoretical considerations and experimental evidence Green and Sawyer considered it reasonable to conclude "that the conversion of CO_2 from the plasma to HCO_3^- in the aqueous humor of the posterior chamber of the rabbit eye probably does not play a significant role in the elaboration or maintenance of the bicarbonate ion concentration in that fluid."

These authors further proposed, on the basis of experimental evidence, that "the bicarbonate ion of the plasma is transported into the aqueous humor as a bicarbonate ion, and that this chemical species is the predominant form in which the transport occurs." And in the following paper (No. VII) Green and Sawyer suggest as a logical corollary of the results of Paper No. VI that "The carbonic anhydrase of the ciliary processes apparently does not participate to any appreciable extent in the elaboration of the bicarbonate ion in the aqueous humor."

The foregoing content is of such fundamental importance to a consideration of the dynamics of aqueous humor formation, that it is indeed surprising that Dr. Maren has apparently not chosen to consider it.

With regard to the mode of action of Diamox in suppressing the rate of flow of aqueous humor and in lowering the bicarbonate ion concentration in that fluid, Green and Sawyer have presented experimental evidence indicating a positive correlation between the suppression of the rate of formation of aqueous humor and the decrease in the rate of transport of bicarbonate ion from plasma, during the first five minutes after injection of the drug. Indeed, the experimental considerations favor the concept that the transport of bicarbonate ion into the eye may be the primary event in the formation of the aqueous humor.

This now brings us to a consideration of the mode of action of Diamox in suppressing the rate of transfer of bicarbonate ion into the eye and the concomitant rate of formation of the aqueous humor. As discussed previously, on the basis that the hydration of CO_2 to H_2CO_3 did not contribute significantly to the elaboration of bicarbonate ion in the aqueous humor, Green and Sawyer concluded that carbonic anhydrase of the ciliary body did not participate to any appreciable extent in the elaboration of the bicarbonate ion in the aqueous humor. Because of the fundamental relationship shown to exist between the rate of elaboration or transport of bicarbonate ion into the eye and the rate of formation of aqueous humor, it seemed reasonable to suggest that the car-

bonic anhydrase also did not participate to any significant extent in the formation of the aqueous humor.

In further support of this Green and Sawyer have pointed out that during the first five minutes after the intravenous injection of Diamox, during which time the rate of transfer of bicarbonate ion and the rate of formation of aqueous humor were markedly suppressed, the carbonic anhydrase activity of the anterior area was not appreciably affected. It is this relationship only that Dr. Maren has questioned!

Dr. Maren's main argument rests upon the contention that while Green and co-workers showed 75 percent inhibition of the carbonic anhydrase activity of the anterior uvea of the rabbit eye 15 minutes after intravenous Diamox and complete inhibition after 30 minutes, they did not measure the activity within the first five minutes. Furthermore, Dr. Maren contends that in association with Dr. Ballintine he had shown "that 10 mg./kg. of Diamox abolished all demonstrable enzyme activity in the ciliary processes 20 minutes after injection and that the concentration of the drug in the anterior uvea was about 100 times greater than that needed for complete inhibition of the enzyme." The implication in Dr. Maren's letter is that the evidence indicates that even within five minutes the enzyme could be considered to have been completely inhibited.

An examination of the data of Green and coworkers reveals that although the activity of the enzyme five minutes after Diamox was not measured, the value obtained by interpolation of the curve was about 20 percent. Even if one assumes that the rates of in vivo inhibition during the first five minutes were faster than indicated, the inhibition would lie between about 20 percent and 60 percent.

As for the findings of Ballintine and Maren referred to above, the results of measurements of carbonic anhydrase activity of isolated ciliary processes in eight experiments showed an average of 23 enzyme units (EU)/gm. of tissue, with a range of from 5.0 to 47 EU/gm. (Average wt. of ciliary processes from one eye, 12 mg.; in seven of the eight experiments the tissues of both eyes were pooled.)

In determining the effect of intravenous Diamox (10 mg./kg.) upon the carbonic anhydrase activity of the ciliary processes, these authors presented data for only two experiments in which, because of insufficient material available, they could not demonstrate the extent of inhibition in the ciliary processes, except that no enzyme activity was detected as far as it was sought. (Is this what Dr. Maren means by his statement "that 10 mg./kg. of Diamox abolished all demonstrable enzyme activity in the ciliary processes 20 minutes after injection, . . ."? The data, however, show that in one experiment, 30 minutes after intraveous Diamox, they could not measure less than the equivalent of 13 EU/gm. and in the other experiment, 20 minutes after injection of the drug, the minimum detectable level was 27 EU/gm. Comparison of these figures with their data for normal animals (average of 27 EU/gm. range from 5.0 to 47) reveals that the minimum detectable enzyme activities after Diamox fell within the range of enzyme activity for untreated normal animals. The authors of course realized that the close correspondence of the results of the two experiments with normal enzyme activity did not permit any valid conclusions with regard to the in vivo activity of Diamox upon the carbonic anhydrase of the ciliary processes.

Having failed to demonstrate in vivo inhibition of carbonic anhydrase of ciliary processes by intravenous Diamox, the authors argue "the reasonable possibility that virtually complete inhibition of carbonic anhydrase is achieved in the ciliary processes," since they have found about 100 times as much Diamox as needed for "complete" in vitro inhibition. Circumstantial evidence of this nature is, at best, only suggestive of possible enzyme inhibition by the presence of the inhibitor substance, provided the inhibitor comes into contact with the intracellular enzyme. These considerations were

understood by the authors who were careful to state in point No. 4 of the summary of their paper that " the amount of Diamox present in iris and ciliary processes after intravenous administration of Diamox (10 or 20 mg./kg.) is enough to inhibit almost completely carbonic anhydrase activity, if the inhibitor reaches the enzyme."

It is obvious, therefore, that Ballintine and Maren have not convincingly demonstrated inhibition of carbonic anhydrase activity of the ciliary processes 20 or 30 minutes after the intravenous injection of Diamox. Therefore, any implication that the enzyme may have been inhibited completely within five minutes of the injection of Diamox is not supported by the data.

The above facts, I submit, negate the statement made by Dr. Maren in his letter that "they (Green and Sawyer) ignore other work (Ballintine and Maren) which shows complete inhibition of the enzyme in the same species (rabbit) using the same drug (Diamox)."

(Signed) Harry Green, Ph.D.
Philadelphia, Pennsylvania.

BOOK REVIEWS

ELEKTROPHYSIOLOGIE DES GESICHTSSINNS. By Wof Müller-Limmroth, Dr. Med., Dozent, Institute of Physiology, University of Münster, West-Germany. Berlin, Springer-Verlag, 1959. 331 pages, 94 figures. Price:59 DM.

Neurophysiology has undergone an almost explosive development during the past 15 or 20 years. Granit's *Sensory Mechansims of the Retina* (reviewed in THE JOURNAL, **30**: 1446 [Nov.] 1947) provided a comprehensive review of visual neurophysiology up to 1947. Müller-Limmroth has now undertaken the formidable task of collecting the overwhelming amount of data published since Granit's book. He has presented these recent advances together with knowledge from an earlier era and thus created a book that covers the entire field of visual neurophysiology from its very beginning to date.

The first two major sections of the book deal with the anatomy of the visual pathways and the different steps in the transformation of radiant energy into nervous activity. The author then devotes approximately one half of the book to theoretical and practical aspects of the electroretinogram. Among the theoretical topics discussed is an interesting section on the origin of the electroretinogram. The fairly recently developed intraretinal microelectrode technique has added much knowledge in this field. For those who wish to use the electroretinogram in clinical ophthalmology enough attention is given to the influence of recording methods upon amplitude, shape, stability, and so forth, of the response to be of practical help. The electroretinogram section concludes with a discussion of clinical conditions in which the electroretinography may be helpful to the ophthalmologist.

The last major section of the book deals with single retinal ganglion cells, the optic nerve, the lateral geniculate bodies and the visual cortex. Here the classic dispute among physiologists—the mechanisms of color vision—has rightly received generous attention.

In many respects this publication duplicates the section on Vision in the recently published *Handbook of Physiology* (reviewed in THE JOURNAL, January, 1960). Nevertheless, Müller-Limmroth's book will be welcomed by many ophthalmologists and neurophysiologists. One of its valuable features is the all inclusive bibliography (a total of 2,400 references!), and an extensive, well-organized subject index. One must be grateful to the author for the seemingly superhuman amount of work involved in writing this book.

Christine Enroth, M.D.

FUNDUS OCULI. By Hector M. Nano. Buenos Aires, Editorial Universitaria, 1958, edition 3. 748 pages, 312 illustrations, 105 in color, 58 tables, indices. Price: not listed.

The jacket of this book gives the impression that it covers a Spanish atlas of the fundus. Although there are numerous color plates of diseases of the fundus of the eye, this text is actually a very detailed treatise on vascular diseases of the retina. It is apparently an enlarged third edition, which speaks well for its popularity in Spanish-speaking countries.

There are 18 chapters, of which the first two deal with papilledema and optic atrophy. The next 12 chapters are devoted to vascular diseases of the retina. There follow three chapters on the retinopathies of pregnancy, diabetes and blood dyscrasias and the final chapter deals with diseases of the choroid.

The textual material is detailed and comprehensive, and black and white photographs excellent. Some of the color photographs are a little flat which may be due to the matte paper used. Some of the tables are helpful, but many of them are simply the textual material rearranged into tabular form. The most valuable portion of the book is probably the extensive bibliography at the end of each chapter. The author has obviously made an effort to cover the literature completely. A minor objection is the chauvinistic division of each bibliographic list into "national" and "foreign" references. The book is recommended to Spanish-speaking ophthalmologists. David Shoch.

THE CAUSE OF BLINDNESS IN THE NETHERLANDS (Blindheidsoorzaken in Nederland). By J. Schappert-Kimmijser. Assen, Van Gorcum & Co., 1959. 137 pages, paperbound. Price: Fl. 9.90.

The Netherlands Association for the Prevention of Blindness promoted this study in order to formulate more definite guiding principles for its program. The significance of the data presented is qualified by the circumstance that the criteria of blindness and the classification of its causes vary now from country to country. An internationally acceptable definition of blindness is yet to be attained. The author, using the standard of 10/200 acuity or less and a visual field of 10-degree radius or less, found in the Netherlands 6,000 blind persons or 55 per 100,000. In all age groups the number of blind males exceeded the number of females—a fact obvious also in American statistics. In two thirds of the children the blindness was of genetic origin. In the Netherlands the chief cause of blindness is tapetoretinal dystrophy in which is included retinitis pigmentosa and Leber's optic atrophy. Glaucoma comes second, and uveitis third. His figures indicate a decrease in blindness from infectious diseases (gonorrhea, syphilis, tuberculosis, and measles) and an increase from trauma and methyl-alcohol poisoning. Making allowance for differences in classification, the causes of blindness in Western countries are similar, but in Eastern countries much blindness is caused by conditions unknown in the occident. James E. Lebensohn.

PRINCIPLES OF DISABILITY EVALUATION. By Wilmer Cauthorn Smith, M.D. Philadelphia and Montreal, J. B. Lippincott, 1959. 204 pages and index. Price: $7.00.

Much of the detail of this volume holds little of interest or importance to the ophthalmologist and for a consideration of the evaluation of visual loss the reader is referred to the Special Committee Reports obtainable from the American Medical Association. The first three sections of the book do contain much of interest in the way of general philosophy and suggestions as to the examination of the patient with an industrial disability. The position of the expert witness in medicolegal testimony is well covered, with excellent advice as to his conduct on the witness stand: "The entire aim of the medical witness should be that of carefully and clearly presenting his opinion, along with every finding and consideration on which it was based, permitting and co-operating in the examination providing further exposition of this material and preventing distortion and confusion from being introduced." William A. Mann.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Cairns, J. E. **Normal development of the hyaloid and retinal vessels in the rat.** Brit. J. Ophth. **43**:385-393, July, 1959.

Despite the extensive use of the rat as a laboratory animal, there is no adequate description of development of the retinal and the hyaloid vessels in the literature. This study has been carried out to provide such data. Animals varying in age from one to 22 days were killed and the retinas removed after 24 hours in formol saline.

At one day of age the hyaloid consisted of a central leash passing through the vitreous to the lens as well as a group of vessels closely applied to the surface of the retina. Both these groups terminated at the lens where they formed a dense meshwork, the tunica vasculosa lentis. This arrangement persists until the fifth or sixth day when about half of the vessels disappeared while the other half became vestigial by the tenth or eleventh day.

The central artery and vein each have six branches and grow rapidly out from the disc; by the eleventh day the superficial system of retinal vessels is adult in form and extent. The deep retinal vessels begin to develop about the ninth day and seem to be fully developed by the fifteenth day. (8 figures, 4 references)

Morris Kaplan.

François, J., Neetens, A. and Collette, J. M. **Vascularization of the optic radiation and the visual cortex.** Brit. J. Ophth. **43**:394-407, July, 1959.

The vascularization of the optic radiation and the visual cortex has been studied by injection of contrast media numerous times and these studies have somewhat changed the concepts of the anatomy of these regions. The optic radiations originate from the carrefour (crossroads) where also are found the termination of the optic tract, the external geniculate body, the primary ganglionic optic center and the Wernicke field. The vascularization of this area is dependent on three arteries: 1. the anterior choroidal which is a terminal artery supplying many branches and collaterals to the anterior part of the radiation, 2. the middle cerebral artery has two important groups of branches, the central perforating branches and the cor-

tical arteries and it supplies the central areas of the radiation and its branches intermingle freely with those of the anterior choroidal artery, and 3. the posterior cerebral artery supplies the posterior and terminal parts of the radiation with many branches and collaterals. There are frequent and very important anastomoses between branches of the middle and posterior cerebral arteries.

The arterioles and precapillaries exhibit a typical architectural pattern in that they never anastomose and remain outside the radiation following the course of the nerve fibers fanwise. The carrefour region includes capillary formations with a typical cellular network radiating like the spokes of a wheel. (16 figures, 21 references)

Morris Kaplan.

Gregersen, E. **The tubular tissue spaces surrounding the endothelial channels of the human iridic vessels. Histologic and postfetal development of the tissue spaces.** Acta ophth. 37:199-208, 1959.

In adults the iris vessels (the endothelial channel and its possible media) are frequently situated in the center of a cylindrical tissue space which is formed by a very strong adventitia. Thus the vessel proper can move freely within the lumen of the outer structure. This arrangement allows the blood to flow freely in the vessels even though they are constantly exposed to kinking by the pupillary movements. The outer tube is denser than the surrounding iris stroma. Killed cocci of a diameter of 0.5 to 1 μ do not penetrate into the tubular spaces, but Dextran molecules of a diameter of about 5 mμ pass the walls. In infants the iris vessels are surrounded by some tissue condensation but this is not true of the tubular spaces seen in adults; the latter develop during childhood. The large diameter of the tubular spaces in adults may be an artefact and in vivo they may be only potential spaces. (3 figures, 20 references) John J. Stern.

Hamburg, A. **Some investigations on the cells of the vitreous body.** Ophthalmologica 138:81-107, Aug. 1959.

The morphologic and staining properties of the cells of the vitreous were studied in eyes of cattle, pigs, cats, rats, chickens, pigeons and sharks by various methods. Teased preparations were obtained by snipping off portions of the peripheral vitreous of fresh eyes and examined directly with a phase microscope or, after staining with methylene blue, with a light microscope. Histologic sections of animal eyes were prepared by a method entailing practically no shrinkage of the vitreous. The author's studies also included the use of vital dyes with paracenteses for the purpose of opening the blood-aqueous barrier.

The findings obtained by these methods characterized the cells of the vitreous as histiocytes originating from the adventitial cells of the papillary vessels, from the ciliary body, and most probably from the retina proper. The normal function of these cells may be that of participation in the anabolism of the vitreous substance. In a number of diseases of the human eye the vitreous cells have been shown to play distinctive parts. (27 figures, over 50 references)

Peter C. Kronfeld.

Unger, H. H. **The anatomy of the chamber angle,** Klin. Monatsbl. f. Augenh. 135: 161-183, 1959.

For purposes of illustration of the topographic relations of the chamber angle, gonioscopic landmarks such as the ciliary body band, the scleral spur, and the trabeculum are correlated with histologic studies of these tissues. Tangential sections were preferred in order to relate these structures as closely as possible to their gonioscopic appearance. The histologic components of the trabeculum are described and illustrated in great detail. In the second part of this paper the author

performed a number of histologic function tests on freshly enucleated eyes. He could confirm histologically that pilocarpine causes actual narrowing of the angle recess and atropine widens it. The circular portion of the ciliary muscle which is adjacent to the uveal wall of the chamber angle shifts the iris root forward and outward towards the corneoscleral wall under the influence of pilocarpine and accommodation. Accommodation results in abduction of the scleral spur which in turn causes unfolding and stretching of the filtration lamellae of the trabecular meshwork. This contributes to narrowing of the chamber angle as well as to dilatation of the transtrabecular drainage system and thus facilitates outflow. (16 figures, 1 table, 106 references)

Gunter K. von Noorden.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Postic, S. **Histologic aspect of the ciliary ganglion in trachomatous individuals.** Arch. d'opht. **19**:402-408, June, 1959.

The author compared the ciliary ganglion in a single normal individual with the ciliary ganglia of three trachomatous individuals with severe Stage IV cicatrization. He noted severe degenerative changes in the ganglia from the trachomatous individuals that he did not find in the ganglion from the healthy control. He noted particularly the multiplication of neuroglia cells to the detriment of the nerve cells. He attributes these changes to the toxic action of trachoma virus which he considers to be an neurotropic virus.

P. Thygeson.

Scardovic, C. and Mulgaria, A. **Effect of treatment with cortisone upon allergic reaction following experimental tuberculosis in rabbits.** Rassegna ital. d'ottal. **28**: 197-203, May-June, 1959.

Six rabbits of the same age, weight and condition were studied; 15 days after the injection of 0.08 mg. of tuberculin all six showed intense reactions. This was followed by a fibrinous exudate into the anterior chamber of two of the animals. One eye was removed and showed a specific granulomatous lesion. To prove that the reaction was specific all rabbits were given cortisone and they responded promptly. Autopsy showed very little difference between treated and untreated animals. It is concluded that the administration of hydrocortisone in allergic animals brings about, in a degree, a blockage of the effect of the tuberculin—a Koch phenomenon. E. M. Blake.

Schwab, F. **Studies on the antibody content of the cornea after local antibiotic medication.** Arch. f. Ophth. **161**:51-63, 1959.

There was a significant difference in the agglutinin and precipitin content of the cornea after subconjunctival penicillin injection in rabbits which had been previously immunized with typhus vaccine. (2 tables, 24 references)

Edward U. Murphy.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Bock, J., Bornschein, H. and Hommer, K. **The survival time of the a-wave in the human electroretinogram.** Arch. f. Ophth. **161**:6-15, 1959.

The ERG was recorded in six eyes during and after enucleation. The positive components were completely gone in two minutes while the negative component (a-wave) persisted up to 30 minutes. These results are discussed in relation to retinal ischemia. (5 figures, 1 table, 21 references)

Edward U. Murphy.

Boros, B. and Takats, I. **Comparative studies with parasympathicolytic agents on the isolated iris sphincter.** Arch. f. Ophth. **161**:32-39, 1959.

The effect of Doryl was noted on the rabbit iris sphincter in vitro after contact with atropine, homatropine, and scopolamine in various dilutions. A comparison was made of the concentrations of these agents necessary to produce the same effect. (7 figures, 11 references)

Edward U. Murphy.

Doden, W. and Boker, W. **The particle size of drugs incorporated in ophthalmic ointments.** Klin. Monatsbl. f. Augenh. **135**:305-347, 1959.

The size of particles of drugs incorporated in ointments is of importance for the compatibility as well as for the effectiveness of the preparation. The available literature contains not only scant data on this subject, but also does not give directions as to the permissible maximal particle size in ophthalmic ointments. The authors undertook, therefore, an extensive study of commercially available ophthalmic ointments and of those prepared according to prescription by local pharmacies. The study revealed that 14 out of 94 brands contained non-visible drug particles. In the remaining group of 80 brands, particles bigger than 50μ were found in 43 brands, and 21 brands contained particles as large as 100 to 400μ. Eight prescriptions were filled by local pharmacies. Four of the prepared ointments contained drug particles of over 50μ in size. An experiment performed in one pharmacy revealed that large drug particles repeatedly pass the type of ointment grinder customarily used by pharmacists. The mechanically irritating effect of ointments containing large particles is shown in rabbits.

The authors conclude that the technical devices used by pharmacists for the preparation of ophthalmic ointments are not as efficient as those used in the drug industry. Industrial grinding of the effective substances into finest powders is recommended in order to facilitate preparation of homogenous and non-irritative ophthalmic ointments by the local pharmacists. Furthermore, it is suggested that the permissible maximal particle size in ophthalmic ointments be limited to 50μ. It is the main purpose of this study to emphasize this fact in view of the pending new edition of the D.A.B. (German Pharmacopeia). (44 figures, 11 tables, 48 references)

Gunter K. von Noorden.

François, J., Neetens, A. and Collette, J. M. **Experimental hypertension of the ophthalmic artery and ocular hypertension. (Preliminary report).** Acta ophth. **37**:395-403, 1959.

If the ophthalmic artery is perfused under a pressure of at least 350 mm. Hg while simultaneously injecting thorotrast into the short posterior ciliary artery under a pressure of 200 mm. Hg, then the eyeball hardens and microradiography shows absence of thorotrast at the level of the lamina cribrosa. If the anterior chamber is punctured, if the pressure of perfusion of the ophthalmic artery is reduced, or the pressure of injection into the short posterior ciliary artery is increased, then microradiography shows the vascular system of the lamina cribrosa to be well filled with thorotrast. If the pressure of injection into the short posterior ciliary artery is reduced while the pressure of perfusion of the ophthalmic artery is maintained at 350 mm. Hg, then no thorotrast is demonstrable in the lamina cribosa. (9 figures)

John J. Stern.

Oksala, A. and Lehtinen, A. **Comparative studies on the electrical conductivity of aqueous humour, vitreous body, cornea and sclera.** Acta ophth. **37**:388-394, 1959.

The electrical conductivity in ox eyes was studied with an audio-frequency generator, a V-T voltmeter and a micrometer with platinum electrodes. The conductivity values in the aqueous and vitreous were $113 \pm 10 \times 10^{-4}$, in the cornea

$26 \pm 3 \times 10^{-4}$, in the sclera near the limbus $19 \pm 3 \times 10^{-4}$, in the sclera near the equator $12 \pm 2 \times 10^{-4}$, and in the sclera near the optic nerve $24 \pm 5 \times 10^{-4}$ (ohm^{-1} cm^{-1}). Measurements of the conductivity of the living eye may become a useful clinical method. (2 figures, 11 references)

John J. Stern.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Rydberg, H. **Optical interference as the basis of the mechanism of vision.** Acta ophth. **37**:305-324, 1959.

A new theory of color vision is presented which is based on the fact that the pars photopica of the retina consists of a pile of uniform double layers which must act as an interference color filter. This double layer consists of alternating protein lamellae and extremely thin lipoid lamellae which have different refractive indices. Its thickness varies in two ways, 1. random variation within "clusters" of cones which will be revealed by microstimulus phenomena, and 2. a successive decrease of thickness outwards from the fovea which is displayed in variations of the color fields. The light absorption, which is responsible for the energy transformation from light to nerve impulse must occur within and by means of the colloidal and ultramicroscopic structures of the protein lamella because the cones contain extremely little photosensitive pigment. Each cone reacts only to the one, two, or three spectral colors which are able to form standing waves in its protein lamella. This implies that the thickness of the lamellae must be an integer multiple of the half-wave lengths of these colors. The chemical, and probably also the first nerve reaction, occur in the protoplasm in the antinodes of the standing waves. The rhodopsin of the rods is the main light absorber and mediates the energy transfer to the rod protoplasm because the rods contain great quantities of this photosensitive pigment, but their lamellae are too thin to enable standing waves to form. (6 figures, 40 references)

John J. Stern.

5

DIAGNOSIS AND THERAPY

Aczel, G. **Modified intermarginal suture.** Klin. Monatsbl. f. Augenh. **135**:425-427, 1959.

A modification of the conventional intermarginal lid suture is described which has the following advantages: the knot is placed beyond the line of the lashes and does not irritate the eye, the suture does not cut through, and entropion cannot develop. (7 figures)

Gunter K. von Noorden.

Brueckner, R. **Longitudinal gerontology: ophthalmological methods.** Ophthalmologica **138**:59-75, July, 1959.

The paper concerns itself with the ophthalmological parameters and the methods for their measurement employed in a broad longitudinal study of the aging process in man which was started in Basle, Switzerland in 1954. Every two years a large group of clinically normal individuals is subjected to a battery of physiologic, medical, psychologic and ophthalmologic tests. The examiners are well aware of the importance of uniformity in the test procedures as well as in the recording of the results. The ophthalmologic methods are therefore described in considerable detail. (4 figures, 7 references)

Peter C. Kronfeld.

Da Luz. B. **Modern concepts in orthoptics.** Rev. brasil. oftal. **18**:219-224, Sept., 1959.

The author feels rather strongly that the reason why many ophthalmologists are sceptical of orthoptics is due primarily to the ophthalmologists themselves. They see a small child with a strabismus and

instruct him to return when he is older. When the child returns one to four years later it is almost impossible to obtain any good results with orthoptics and therefore the ophthalmologists feel that orthoptics is of no value. The author emphasizes the need for using orthoptics at the earliest possible age; no age is too young.

Walter Mayer.

Ehrich, W. and Piening, O. **Decisive factors for lasting results of pleoptic treatment.** Klin. Monatsbl. f. Augenh. **135**:394-400, 1959.

The authors have re-examined 107 out of 203 patients who had had pleoptic treatment with the Bangerter method. Of the total of 203 cases, 35 percent were considered to be therapeutic failures, 27.1 percent showed some degree of recurrent amblyopia, 15.3 percent had permanent improvement, and 10.3 percent were classified as having lasting successful therapeutic results. This study revealed also that fixation behavior and duration of treatment is of no significance for the prognosis of the final result. The extent of the achieved binocular coordination as well as the angle of squint are of decisive influence on the outcome of treatment. (6 figures, 1 table, 18 references)

Gunter K. von Noorden.

Eisum, E. F. **Tangent screen illumination.** Acta ophth. **37**:386-387, 1959.

Four Comptalux-Flood lamps (made by Philips) give a uniform illumination of about 450 to 650 lux on a tangent screen when mounted 3.5 meters away.

John J. Stern.

Eisum, E. F. **A demagnetiser for surgical instruments.** Acta ophth. **37**:381-385.

A simple instrument is described which consists basically of a plastic tube covered with enameled copper wire windings, into which steel instruments are introduced and withdrawn for complete demagnetization. (4 figures) John J. Stern.

Gessler, A. **Relations between early lipoid arcus of the cornea and circulatory disorders of the heart.** Ophthalmologica **138**:118-133, Aug., 1959.

Ophthalmological examinations of patients under 55 years of age with gross lipoid arcus of the cornea revealed 1. marked predominance of men over women, 2. a hereditary factor, and 3. a high incidence of previous eye diseases or exposure, over long periods, to local irritating, chemical or physical factors. There was no definite correlation between early lipoid arcus and disorders of the coronary vascular system. (4 tables, 25 references) Peter C. Kronfeld.

Millan Hernandez, Jose. **Chisel and hammer in dacryocystorhinostomy.** Arch. Soc. oftal. hispano-am. **19**:364-367, May, 1959.

The author regrets that the dependence on electric driven instruments leads to abstention from the operation when such instruments are not available. He describes the technique of using a hammer and chisel, which leads to just as satisfactory results. (2 figures, 2 references)

Ray K. Daily.

Queiroga, G. **Simplified fundus photography.** Rev. brasil. oftal. **18**:205-210, Sept., 1959.

The author describes his method of combining in one unit the Bausch and Lomb binocular ophthalmoscope and a single reflex camera, which allows one to observe the fundus and at the same time take pictures of the fundus in a rather easy manner. (4 figures, 3 references)

Walter Mayer.

Queiroga, G. **Slitlamp microscopy and photography.** Rev. brasil. oftal. **18**:211-216, Sept., 1959.

The author uses a diffuse illumination and the slit from any type of slitlamp from which the microscope has been re-

moved and substituted by a single reflex camera. (3 figures, 5 references)

Walter Mayer.

Ricci, A. and Canossa, A. **The indirect ophthalmoscope of the Eye Clinic in Geneva (Switzerland).** Ophthalmologica **138**:10-12, July, 1959.

A monocular indirect ophthalmoscope with a built-in, line-voltage fed 15 watt bulb has proved thoroughly satisfactory for general use and particularly during retinal detachment operations. (2 figures)

Peter C. Kronfeld.

Vannas, S. and Larmi, T. **The use of collagenous suture material in surgery of the cornea and sclera.** Acta ophth. **37**:371-380, 1959.

The fine collagenous fibers in the rat tail, sterilized in ethylene oxide, make suitable corneal and corneoscleral suture material. (5 figures, 19 references)

John J. Stern.

6

OCULAR MOTILITY

Falbe-Hansen, I. and Gregersen, E. **The prognosis for disturbances in ocular motility following trauma to the head.** Acta ophth. **37**:359-370, 1959.

Fifty-eight patients with disturbances of ocular motility after craniocerebral trauma were studied. Of the supranuclear disturbances seven cortical paralyses resolved within one to three weeks with the recovery of consciousness. Of six brain stem paralyses five resolved completely after about two months; the sixth patient showed a very slight nystagmus on lateral gaze after nine years. In 18 patients with isolated paralyses of the oculomotor nerve (nine were both total internal and external, and nine were partial external paralyses) the paralysis disappeared in 14 cases (six total and eight partial). One isolated superior oblique paralysis was permanent. All isolated abducens paralyses (15) resolved. In 10 cases of mixed or multiple paralyses, five persisted. In the patients with nuclear or infranuclear paralysis the recovery time was two to nine weeks. The average time of observation was two and one half years. (1 figure, 23 references)

John J. Stern.

Lapresle, J. and Jarlot, J. **Ocular myopathy. Contribution to the study of progressive external ophthalmoplegias.** Arch. d'opht. **19**:384-396, June, 1959.

The authors refer to recent literature indicating that progressive external ophthalmoplegia is not due to degeneration of oculomotor nuclei as previously thought, but rather to dystrophic changes in the muscles themselves. They report three cases of the disease in which histologic examination showed them to be ocular forms of progressive muscular dystrophy. The first patient, a man with onset of his trouble at age 40, had ptosis and slight exophthalmos but no lesion except of the eye. Biopsy of the right external rectus muscle 15 years after onset revealed dystrophic lesions. In the second case, a male with onset of difficulty at age 24, had involvement of his arms as well as typical ocular involvement. Three muscle biopsies, including one of the left external rectus, showed typical dystrophy. Ten years after onset difficulty in walking developed. The third case, a woman with onset of her eye difficulty at age 44, later developed widespread sketal muscle changes. Biopsy of skeletal muscles showed typical dystrophic changes, but biopsy of an eye muscle was not accomplished. The authors conclude by stressing the importance of biopsy in the diagnosis of these cases.

P. Thygeson.

Levy, J., Saraiva, S., de Carvalho, C and Andrada, J. **Ophthalmoplegic progressive muscular dystrophy.** Rev. brasil. oftal. **18**:227-235, Sept., 1959.

The authors present a case of progressive nuclear ophthalmoplegia and review briefly the latest literature on this subject. They feel that this disease is an ocular manifestation of a progressive muscular dystrophy, which seems to be corroborated by histologic and electromyographic studies of the extraocular muscles. (7 figures, 5 references) Walter Mayer.

Sachsenweger, R. and Lukoff, L. **Congenital facial asymmetries as a cause of strabismus.** Klin. Monatsbl. f. Augenh. **135**:388-394, 1959.

Concomitant strabismus was present in 13 out of 30 patients with facial asymmetries which were birth complications or intrauterine hereditary deformities. Asymmetry of muscular function as well as anisophoria and its combination with other squint-producing factors, such as impairment of motor fusion centers, are thought to be active in the development of strabismus. Most probably such influences are only significant in early childhood when binocular coordination has not attained its final stability. Facial hemiatrophy occurring at a later age rarely causes concomitant strabismus. (26 references) Gunter K. von Noorden.

7

CONJUNCTIVA, CORNEA, SCLERA

Fazakas, S. **Summarizing report on mycotic superinfections in diseases of the lid borders, conjunctiva and cornea.** Ophthalmologica **138**:108-118, Aug., 1959.

Fungi as secondary pathogens causing superinfection were identified in a variety of primary eye diseases (trachoma, phlyctenular disease, dendritic keratitis, serpentic ulcer, catarrhal ulcer and ulcerative blepharitis). The effect of the secondary mycosis may be aggravation or mitigation of the primary disease. (16 references) Peter C. Kronfeld.

Galvez Montes, Jose. **A family with macular corneal dystrophy.** Arch. Soc. oftal. hispano-am. **19**:319-322, May, 1959.

This is a brief report of a case of macular corneal dystrophy in a man 42 years old, and an analysis of his familial tree which revealed four affected members in three generations. The differential diagnosis between the two forms of Groenouw's dystrophy is discussed and the prognostic implication of the differentiation is pointed out. In the macular form the opacities are diffuse, extend to the corneal periphery, and vision is severely impaired, whereas in the granular form the opacities are circumscribed, the periphery is left transparent and the impairment of vision is less intense. Generally the epithelium remains intact in the macular form and the corneal sensitivity is diminished whereas the opposite is the case in the granular type; this differential feature is variable and not always reliable diagnostically. The heredity in the granular form is dominant in type, and that of the macular form is recessive; the prognosis for transmission of the affection is therefore also different. (1 table; 6 references) Ray K. Daily.

Pau, H. and Baumer, A. **Corneal chloroquine deposits.** Klin. Monatsbl. f. Augenh. **135**:362-377, 1959.

Slitlamp examination was done in 26 patients who had received chloroquine for various lengths of time for chronic degenerative joint diseases and lupus erythematosus. Epithelial deposits were demonstrated in all patients who had taken the drug for two months or longer. The deposits were of a brownish or yellow-brown color, and more frequent in the area of the palpebral fissure. Histologic examination of the denuded epithelium was made in one case and revealed punctiform intracellular inclusions, sparing the cell nucleus. The condition is fully reversible; after discontinuation of the drug

the cornea clears. (15 figures, 26 references) Gunter K. von Noorden.

Rogers, A. **The cornea.** Rev. brasil. oftal. 18:253-268, Sept., 1959.

The author summarizes briefly the most important anatomic and physiologic characteristics of the cornea. (10 references) Walter Mayer.

Sharkovsky, J. A. **Fungal keratitis (corneal aspergillosis).** Vestnik Oftal. 1:26-30, Jan.-Feb., 1959.

Three cases of corneal aspergillosis are reported, all associated with slight trauma to the eye. The eye was lost in two cases and retained in one. All of the patients were given local and systemic antibiotics, and none of them had steroid therapy. One of the eyes which was lost had been given paracentesis and irrigation of the anterior chamber with penicillin. The author remarks on the extreme rarity of this diagnosis but believes that it will be made more frequently when it is kept in mind. (2 figures 17 references) Victor Goodside.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Agarwal, L. P. and Malik, S. R. **Postoperative iris prolapse, its cause and management.** Ophthalmologica 138:133-137, Aug., 1959.

Postoperative iris prolapse occurs in about 2 to 4 percent of the cataract operations performed in the Department of Ophthalmology of the G.S.V.M. Medical College in Kampur, India. These prolapses have been treated by partial excision, reposition of the pillars and resuturing of the wound. The visual end-results in these cases have been significantly worse than in cases with uncomplicated postoperative course. In an endeavor to improve their visual results the authors have repaired 50 iris prolapses by 1. removing the corneoscleral suture nearest the prolapse, 2. allowing the vitreous which was invariably present in the anterior chamber to prolapse, 3. reposition of the iris without excision, 4. irrigation of the anterior chamber with saline solution, 5. resuturing of the wound, 6. air injection into the anterior chamber, and 7. subconjunctival injection of 100,000 units of penicillin. In nine of the 50 cases iritis developed on the fourth postoperative day and was treated with hydrocortisone topically and butazolidine intramuscularly. The visual end results in this series of cases were decidedly better than those in a control series of iris prolapses treated by the "older" method of partial excision and resuturing of the wound. (2 tables) Peter C. Kronfeld.

Sandvig, K. **Central areolar choroidal atrophy. A report on four cases.** Acta ophth. 37:325-329, 1959.

A description of four cases, illustrated by excellent fundus photographs, with a discussion of differential diagnosis and the etiology is provided. (6 figures, 12 references) John J. Stern.

Schmitzer, G., Lichtenberg, R., Lasco, F., Pantzer, L., Norz, L. and Nicolesco, M. **The treatment of iridocyclitis with hard roentgen rays.** Ophthalmologica 138:138-145, Aug., 1959.

The principle underlying the technique of radiation employed by the authors is that doses of hard X-rays which are effective in the treatment of iridocyclitis, do not cause radiation cataracts. The authors obtain this hard radiation by the use of a current of 180 to 200 kilovolts and a 0.5 mm. copper filter at a distance of 40 cm. between the tube and the eye. In cases of acute iridocyclitis the individual dose is 30 to 60 r, repeated after 24 to 48 hours. Depending upon the response of the eye, these treatments are continued until the

inflammatory symptoms have completely subsided. Stubborn cases may require as much as 500 to 700 r.

In chronic iridocyclitis a total amount of 300 to 400 r, given in individual doses of 60 r every two days, is usually sufficient. This course of treatment is followed by a rest period of three to four weeks during which the effects of the radiation gradually come to the fore. If there is no perceptible change in the clinical manifestations of the disease a second course of X-ray therapy may be given four to five weeks after the first course. If the first course results in aggravation of the clinical symptoms, therapeutic methods other than radiation should be tried.

Between 1950 and 1957, 958 cases of iridocyclitis were treated by the method described. The results are reported as cures in 62 percent, improvements in 27 percent, and aggravation in 1 percent, with the remaining 10 percent of the cases having been uninfluenced. Mydriatrics were used topically. No immediate or late injurious effects of the radiation and particularly no radiation cataracts were observed. (1 reference) Peter C. Kronfeld.

9

GLAUCOMA AND OCULAR TENSION

Auricchio, G. and Wistrand, P. **The osmotic pressure in the aqueous humour of rabbits with congenital glaucoma.** Acta ophth. **37**:340-343, 1959.

In rabbits with congenital glaucoma the normal excess of osmotic pressure in the aqueous as compared to blood plasma is abolished. The drop of the pressure which follows a carbonic anhydrase inhibitor is much delayed. (2 figures, 19 references)

John J. Stern.

Cramer, F. K. and Lamela, N. A. **Scleral rigidity and intraocular pressure, with special reference to applanation tonometry.** Arch. oftal. Buenos Aires **33**:19-26, Jan.-March, 1959.

Current notions on scleral rigidity and the manner in which this factor may affect and occasionally vitiate the results of Schiötz tonometry, are summarized and discussed. The Goldmann applanation tonometer is described, less from the point of view of its theoretical basis than from that of its practical application. Particular consideration is given to Goldmann and Schmidt's excellent procedure for the clinical determination of scleral rigidity with the aid of the former's tonometer, a standardized Schiötz tonometer, and Friedenwald's well-known nomogram. No mention is made, however, of the ingenious method devised by Schmidt (cf. Schmidt, Th.: Fortschritte in der Differential-tonometrie, Klin. Monatsbl. f. Augenh. 129:196, 1956, and Der "Differenzwert" ein Mass für die Augapfelrigidität, ibid. 131:195, 1957) for the correction of Schiötz tonometer readings in a given eye by means of the so-called "difference values." (1 table, 1 graph, 3 figures, 16 references)

A. Urrets Zavalia, Jr.

De Rezende, C., de Carvalho, C., Pompeu, C. and Attanes, M. **The ocular hypotensive action of Neptazane.** Rev. brasil. oftal. **18**:191-197, Sept., 1959.

The authors report their findings with the use of Neptazane in glaucomatous patients. They studied this carbonic anhydrase inhibitor in 10 patients with chronic open-angle glaucoma and in two patients with secondary glaucoma. Tonographic studies revealed that there was a marked decrease in the aqueous flow and therefore a lowering of the ocular tension was obtained. (3 tables, 2 references)

Walter Mayer.

Illig, K. M. **A new filtering operation for glaucoma.** Ophthalmologica **138**:54-58, July, 1959.

After preparation of a limbus-based conjunctival flap the author makes a

chamber-opening keratome incision about 2 mm. behind the limbus. In this incision he places a short piece of tightly fitting polyethylene tubing and closes the conjunctiva over it. Experiences in four cases with short follow-up periods suggest that the implantation of plastic tube into scleral openings may represent a less traumatic technique of filtering operation for glaucoma than the iridencleisis. (4 figures) Peter C. Kronfeld.

Pillat, A. and Nemetz, U. R. **Iridencleisis in acute glaucoma.** Arch. f. Ophth. 161:40-44, 1959

Iridencleises was performed on 140 patients who were followed two to ten years. In only seven did the pressure rise again and all of these were controlled by cyclodialysis. (2 figures, 21 references) Edward U. Murphy.

Pompeu, C. and de Carvalho, C. **Decrease of the intraocular tension with the use of Cardrase.** Rev. brasil. oftal. 18:199-204, Sept., 1959.

The authors studied the effects of miotics and Cardrase over a short period of time in 16 patients with open angle chronic glaucoma and in two patients with secondary glaucoma. They found that there was a marked decrease in the ocular tension due to a reduction in the aqueous flow. (2 tables, 3 references) Walter Mayer.

Senà, J. A. **Remarks on the technique of iridencleisis.** Arch. oftal. Buenos Aires 33:4-8, Jan.-March, 1959.

If iridencleisis is to be of lasting success in relieving ocular hypertension, the three following requisites ought to be fulfilled: 1. the iris stroma must be thick enough (i.e., non-atrophic) when observed with the slitlamp, 2. the incision should be located 0.5 mm. behind the posterior limit of the limbus as seen after the conjunctiva has been dissected as far anteriorly as its insertion into the corneal tissue; the iris root will then lie immediately behind the posterior edge of the wound, and 3. the section must be made perpendicular to the scleral surface, so that the incarcerated pillars are not submitted to an undue backward deflexion. Though the two latter postulates are undoubtedly sound, one cannot help but wonder whether the former one is of real practical consequence when one thinks of the very good results obtained in narrow-angle (iris-block) glaucoma, where the iris has often become markedly atrophic after one or more acute, congestive attacks. (4 figures, 6 references) A. Urrets Zavalia, Jr.

Serpell, G.: **Provocative tests for glaucoma.** M. J. Australia 2:598-600, 1959.

The following provocative tests are discussed: bulbar pressure test, tonography, water drinking, mydriatic, priscol, darkroom, and reading. Gonioscopy is necessary before performing any provocative test. The author feels that phasic variation records give the most definite information. Ronald Lowe.

Topalis, C. and Roussos, J. **Some considerations on iridencleisis.** Arch. d'opht. 19:397-401, June, 1959.

The authors describe in detail the history of the operation of iridencleisis, its various modifications, and the technique in use at the eye clinic of the Faculty of Medicine of Athens. They describe its use in cases of chronic simple glaucoma, in cases of glaucoma with inflammation, and even in congenital glaucoma. They report on 67 cases, in 53 of which two pillars were incarcerated. In this latter group well-maintained normalization of tension was obtained in 35 cases, whereas in 14 cases in which a single pillar was incarcerated, 11 had a satisfactory result. In 12 cases of the series the ocular tension

remained high while in six there was marked hypotony. The authors conclude that iridencleisis has many advantages over other types of fistulizing operation.
P. Thygeson.

Vannas, S. **Mast cells in glaucomatous eyes.** Acta ophth. **37**:330-339, 1959.

In simple glaucoma no significant elevation of the mast cell content in the iris was found. In closed-angle glaucoma the mast cell content of the iris tissue varied with the phase; during an acute attack their number fell, even down to zero. After the attack, and in the chronic congestive phase, the mast cell count was significantly higher than normal. This increase is probably the expression of a reparative process. (1 figure, 2 tables, 28 references) John J. Stern.

10
CRYSTALLINE LENS

Arrechea, A. and Adrogue, E. **Enzymatic zonulysis.** Rev. oto-neuro-oftal. Sudam. **34**:15-18, Jan.-May, 1959.

The authors present their first experiences with alfa quimotripsine, having used the product manufactured in Spain, and following closely the recommendations of Barraquer, using the 1:10,000 solution in adults and 1:5,000 in younger patients. They conclude that the use of this drug facilitates the extraction of the cataractous lens but they warn that striate keratitis may develop and eyes may remain congested for a long time after the procedure. They also feel that the use of alfa quimotripsine in congenital cataracts must be more carefully evaluated; because of the frequent presence of an adhesion between hyaloid membrane and posterior lens capsule, drops of the drug must be instilled into the eye slowly after the lens has become already somewhat dislocated in order to prevent vitreous loss. (1 table) Walter Mayer.

Bon, W. F. **Lens opacities due to cooling.** Ophthalmologica **138**:34-39, July, 1959.

The author's experimental studies suggest that the response of the lenses of newborn or very young mammals to cooling below body temperature with the development of a reversible type of lens opacification may be dependent on the prevailing concentrations of alpha and beta crystallin. (24 references)
Peter C. Kronfeld.

DaSilva, J. L. L. and Malta, A.S. **Symposium on Pathology of the Crystalline Lens.** Arq. brasil de oftal. **22**:1-49, 1959.

Da Silva, J. L. L. **Heredity, immunology, general status, pathological anatomy, pathogenesis** pp. 1-42.

Various anomalies of the crystalline lens are hereditary in nature, such as congenital subluxation, coloboma, microphakia, spherophakia, and cataract. Congenital cataract as well as postnatal cataracts may be hereditary and are usually dominant in character, although there are instances where the condition is recessive.

The possible relation of immunologic reactions to cataract is interesting. Normal individuals and those with cataract differ greatly in their reactions to intradermal injections of heterologous crystalline protiens. Patients with cataract have been shown to be hypersensitive to these proteins while normal individuals are not. This is confirmed by observations of sensitivity reactions following extracapsular cataract extraction, with the absence of such reactions in intracapsular extraction. A discussion of endophthalmitis phakoanaphylactica completes the section on immunology.

Numerous factors associated with cataract formation are mentioned, such as degenerative diseases, metabolic disorders, dietary factors, toxic reactions and trauma. Photomicrographs of various lens conditions and a discussion of the patho-

logic findings complete the article. (photographs, genalogical charts, 21 references)

James W. Brennen.

Malta, A. S. **Slitlamp indications in cataract surgery.** pp. 43-49.

Biomicroscopy of the lens allows the surgeon to evaluate the lens changes in detail and thereby increases the chance of success in cataract surgery. The author classifies lens changes in three groups: physiologic senile alterations, premonitory signs of cataract, and the lens changes found in cataract. A brief description of the biomicroscopic findings in each of these groups is given. (5 references)

James W. Brennan.

Hammar, H. and Brolin, S. E. **The fluorescence of the eye lens in rats with Myleran cataract: a spectro-photometric investigation.** Acta ophth. 37:344-349, 1959.

The fluorescence of the lens in Myleran-induced cataracts of rats increases by about 10 percent and only a slight shift in the spectral distribution takes place. The same has been found in galactose, tetanic or roentgen cataracts, while naphthalene and diabetic cataracts are characterized by a marked increase in fluorescence. (2 figures, 1 table, 11 references)

John J. Stern.

Junceda Avello, J. **Delayed reformation of the anterior chamber in cataract extraction.** Arch. Soc. oftal. hispano-am. 19:355-363, May, 1959.

After a brief reference to the literature the author states as his own criterion that a delay of 48 hours in the reformation of the anterior chamber be regarded as suspicious and after 72 hours as pathological. The etiology, symptomatology, prognosis and therapy of this complication are discussed. The author holds that the longer the reformation is delayed the greater the probability of late complications, and the more irritable the eye the more urgent is medical or surgical therapy. The author's treatment pattern for delayed reformation of the anterior chamber consists in the suppression of steroids, stimulation of circulation and metabolism, touching the leaking points with the electrocautery or acid; surgical removal of tissues incarcerated in the wound; sutures which penetrate the anterior chamber, and sedatives to eliminate spasm of the orbicularis. In the presence of a vitreous detachment the pattern includes absolute bed rest with head elevated and a binocular bandage, hypertonic solutions intravenously and diuretics orally, miotic drops and ointments, cortisone locally, instillation of air into the anterior chamber, and transcleral evacuation of the subchoroidal fluid. Closed angle glaucoma demands miotics and cyclodiathermy or cyclodialysis, if indicated. Prolapse of vitreous may require air injection into the anterior chamber or a posterior sclerotomy. Iris block is treated with mydriatics followed, if ineffective, by transfixion of the iris.

Ray K. Daily.

Krasnov, M. M. **The method of forced blood evacuation from the suprachoroidal space in the treatment of expulsive hemorrhage.** Vestnik Oftal. 1:30-33, Jan.-Feb., 1959.

Intra-capsular cataract extraction associated with vitreous loss was followed the next day by severe pain in the eye. Although there was no disruption of the wound, the anterior chamber was emptied behind the pupillary area, and the tension elevated. Several days later the anterior chamber had reappeared but the retina remained visible in the pupillary area. The patient was returned to the operating room and five diathermy punctures were made in the sclera at the equator. A notable amount of dark red blood escaped from the puncture wounds. A

needle was introduced into the anterior chamber at the limbus and 3 or 4 cc. of air was injected into the eye. As the air entered the vitreous chamber more blood escaped through the scleral puncture wounds. The retina and choroid were postoperatively noted to be in place, a vitreous haze was in evidence, and there was some of the corneal epithelium. The final visual result three months after the surgery was 0.4. The author suggests that scleral trephination would have been preferable to the diathermy punctures. (12 references) Victor Goodside.

Stucchi, C. and Shapiro, D. **The use of prazine in the preparation for cataract surgery.** Ophthalmologica **138**:146-150, Aug., 1959.

Prazine is a phenothiazene, known in the U.S. under the name of sparine. Combined with atrophine, phenergan and demerol, prazine is used routinely in the University Eye Clinic of Lausanne as a preoperative sedative for cataract surgery. The respective doses of the four agents are adjusted to the patient's age and his cardiovascular status. The results have been satisfactory; the patients are well relaxed and cooperative during the surgery and free from nausea and vomiting after the surgery. (2 references)

Peter C. Kronfeld.

11

RETINA AND VITREOUS

Hommer, K. **The ERG in sector-type retinitis pigmentosa.** Arch. f. Ophth. **161**: 15-26, 1959.

The author studied three members of one family who had the typical changes of retinitis pigmentosa confined to one quadrant of each eye. The ERG was normal and indicated that the apparently unaffected retina was indeed free of degenerative changes; the ERG would consequently not be helpful in the early diagnosis. (5 figures, 17 references)

Edward U. Murphy.

Pau, H. **The plombage operation for retinal detachment.** Ophthalmologica **138**: 12-34, July, 1959.

The principle of Custodis' plombage operation is 1. surface diathermy applied to the sclera overlying the retinal breaks and 2. indentation of this portion of the eyeball wall by means of short plastic rods held in position by strong suture material (supramid). With this method the author and his associates achieved anatomical cures in 84 percent of a series of 302 cases of retinal detachment with satisfactory follow-up. Data pertaining to factors such as age, refraction, location and number of breaks, and possible precipitating factors are given in the form of tables and graphs. A typical operation is described in detail and clearly illustrated (cfr. American Journal of Ophth. 47:565, April, 1959). (9 figures, 38 references)

Peter C. Kronfeld.

Vukovich, V. **The ERG in retinitis pigmentosa with bitemporal field defects.** Arch. f. Ophth. **161**:27-31, 1959.

A case of this disease is described with pigmentation only in the nasal half of the retina. The disc and the retinal arterioles showed typical changes and the ERG was almost entirely extinguished. (3 figures, 1 reference) Edward U. Murphy.

12

OPTIC NERVE AND CHIASM

Busacca, A. **Morphogenesis of the papilla of stasis. Biomicroscopic study.** Arch. d'opht. **19**:361-383, June, 1959.

The author notes that biomicroscopic study has greatly aided ophthalmoscopic study of the papilla by providing stereopsis and optical section. He then pro-

ceeds to analyze the various modifications of the normal and the edematous papilla and illustrates them with charts and figures, some in color. In the differential diagnosis of papilledema, he considers concretions of the papilla and edema propagated from the retina, as in certain cases of hypertension, as well as edema in the course of inflammatory disease of the retina. He concludes by sharply differentiating pure papilledema of stasis from inflammatory involvement of the disc on the basis of biomicroscopic findings. This excellent report is worthy of careful study by all ophthalmologists.

P. Thygeson.

13

NEURO-OPHTHALMOLOGY

Grosch, H. **Pathogenesis of Adie's syndrome.** Klin. Monatsbl. f. Augenh. **135**: 401-416, 1959.

Motor, psychopathologic, and oculopupillary disturbances comprise the syndrome of the mesodiencephalic transitional zone. Functionally the symptoms may be attributed to deficient dissociation between the stages of sleeping and being awake. The observation of tonic pupil and Adie's syndrome in these conditions suggests that in the waking stage persistent hypnogenic factors lead to tonic dysregulations at the receptory initial as well as at the effective terminal link of the animal system. (33 references)

Gunter K. von Noorden.

Palomar Collado, F. and Palomar-Petit F. **Semiologic ophthalmoneurology in two cases of aneurysm of the posterior choroidal arteries.** Arch. Soc. oftal. hispano-am. **19**:323-330, May, 1959.

After a brief review of the cerebral blood vessels, with special emphasis on the posterior cerebral arteries and its branches, the authors report the neuro-ophthalmologic symptoms in two rare cases of aneurysm of the posterior choroidal arteries. Aneurysm of the posterior cerebral artery constitutes but three percent of intracranial aneurysms, and aneurysm of its branches is still rarer. The initial symptoms of the authors' first patient who had an arteriovenous aneurysm of the posterior cerebral artery, were ophthalmic migraine, scintillating scotoma, vasomotor disturbances, nausea, vomiting, and dizziness; as the disease advanced there developed diplopia, papilledema and left-sided constriction of the visual field. In the second case, angiography of the left carotid artery demonstrated an abnormal enlargement in the region of the posterior choroidal artery in a man who was 42 years old. For 24 years the patient had had headache, principally in the occipital region, and repeated episodes of subarachnoid hemorrhage with signs of cerebello-vestibular disturbances, anisocoria, nystagmus on gaze up and to the right, and partial right inferior homonymous quadrant anopsia. (3 figures, 11 references)

Ray K. Daily.

14

EYEBALL, ORBIT, SINUSES

Bleeker, G. M. and Van Ommen, B. **The early treatment of orbital fractures.** Ophthalmologica **138**:40-53, July, 1959.

A sizable series of orbital fractures convincingly demonstrates the advantages of recognition and reduction of the fracture within the first two weeks after the injury. Three groups of fractures may be distinguished: 1. separation of the zygoma from its frontal and maxillary attachment, including the orbital floor to a greater or lesser extent, 2. the blow-out fracture of the orbital floor or medial wall with intact orbital rim, and 3. fracture of any of the orbital walls by a pointed object such as the horn of a cow, without damage to the orbital rim. In order not to overlook any of these fractures, stereo X-ray pic-

tures in a mento-occipital direction should be taken in any case of severe head trauma. (16 figures, 3 references)

Peter C. Kronfield.

Fernandez-Gonzales, Angel. **New techniques in evisceration and enucleation.** Arch. Soc. oftal, hispano-am. **19**:331-351, May, 1959.

After a comprehensive review of the literature on the various techniques of evisceration and enucleation the author describes briefly a technique of implantation of a mass, which consists of the patient's own sclera or preserved cadaver sclera around a plastic sphere. The chief merit of this type of implant is the restoration of muscular attachments. Two cases are reported in which this procedure was used, and in which a satisfactory result was maintained over a period of observation of more than one year in one and two months in the other. In one patient a plastic mass in a preserved cadaver sclera was implanted 19 years after the enucleation because ectropion of the lower lid had developed and the orbit was unable to hold the prosthesis. In the second case the patient's own sclera was used immediately after the enucleation. (5 figures, 30 references)

Ray K. Daily.

Molnar, L. **A case of posterior orbital encephalocele.** Klin. Monatsbl. f. Augenh. **135**:427-431, 1959.

A 24-year-old woman developed unilateral exophthalmos, ipsolateral orbital pain, and visual loss. Skull films revealed marked dilation of the optic canal on the affected side. A tumor of the optic nerve was diagnosed and a Kroenlein procedure carried out. An encephalocele with intradural protusion through the optic canal was found. The patient had had severe hyperemesis gravidarum prior to onset of symptoms. In retrospect the author concludes that the increase of intracranial pressure caused by persistent vomiting had resulted in protusion of brain tissue through a congenitally dilated optic foramen. The literature on orbital encephalocoeles is reviewed. (4 figures, 10 references) Gunter K. von Noorden.

Zeidler, I. and Zetterström, B. **Analysis of cases of venous cavernous angioma of the orbit.** Acta ophth. **37**:350-354, 1959.

One case of retrobulbar cavernous hemangioma in a 25-year-old woman is reported. During induced stasis of the vessels of the head, a decrease in visual acuity and a supernormal ERG were recorded. (2 figures, 7 references)

John J. Stern.

15

EYELIDS, LACRIMAL APPARATUS

Huggert, A. **The treatment of stenosis of the lacrimal canaliculi.** Acta ophth. **37**: 355-358, 1959.

Canalicular stenosis is treated by passing the free end of a polyethylene tube through the upper and lower canaliculi to emerge in the nasal cavity. In 15 out of 20 cases an open passage was achieved. (1 figure, 10 references)

John J. Stern.

16

TUMORS

Davids, Bernd. **The problem of melanoma and glioma development on the basis of comparative growth studies on 43 melanomas and 12 gliomas.** Arch. f. Ophth. **161**:1-5, 1959.

The author speculates on the microscopic and biochemical basis for the origin and growth of both kinds of intraocular tumors. Both seem to have an almost identical early development. (9 refrences). Edward U. Murphy.

Eross, S. **Treatment of intraocular metastatic carcinoma.** Klin. Monatsbl. f. Augenh. **135**:417-424, 1959.

A short review of the literature on this subject is provided. The author recommends irradiation of the posterior portion of the globe; this led to improvement of vision in his patients until death as a result of the primary lesion. The doses ranged from 800 to 5,600 r. (6 figures, 1 table, 37 references)

Gunter K. von Noorden.

Viana, L. **Epibulbar tumors.** Rev. brasil. oftal. **18**:237-250, Sept., 1959.

The author found seven cases of epibulbar tumors among 30,000 patients. Most of these tumors are benign and the great majority are nevi. The biomicroscope may be of great help in determining the change from a benign into a malignant nevus. The cornea was never invaded by these tumors and only at times half hidden behind a tumor arising near the limbus. (31 references). Walter Mayer.

17

INJURIES

Handmann, W. **Burns and regeneration.** Klin. Monatsbl. f. Augenh. **135**:353-361, 1959.

The author reports his treatment of ocular burns. The injuries were caused by various chemical agents. The treatment consisted mainly of immediate lavage with boiled tap water, and subsequent local ultraviolet irradiation. This is followed by high irrigation with an undine and a moist dressing. Four case reports are included. The regeneration of tissues damaged by chemicals is discussed. (2 tables, 52 references)

Gunter K. von Noorden.

Honegger, H. **Experimental investigations on irrigation therapy of fresh lime burns with complex forming solutions.** Klin. Monatsbl. f. Augenh. **135**:347-352, 1959.

Lime burns of various degrees were experimentally produced in rabbit corneas and the eyes were irrigated with a ¼, 1, or 4 percent solution of the sodium salt of EDTA (ethylene-diamine-tetra-acetic acid). Subsequently, the still soluble amount of calcium was determined by flame photometry. That irrigation with EDTA after lime burns is of distinct advantage in comparison with irrigation with water as was shown by a control group. Other favorable properties of EDTA treatment of lime burns are discussed. In man the author uses a 1-percent solution for fresh lime burns. For corneal dystrophies with calcium deposits, a 1-percent solution without neutralizing additions, and an ointment containing EDTA is recommended. In contrast to reports of others, the author does not denude the corneal epithelium prior to application of EDTA. (1 figure, 18 references) Gunter K. von Noorden.

19

CONGENITAL DEFORMITIES, HEREDITY

Thier, C. J. **Syndromes within the scope of mandibulo-facial dysplasias. Malformations in the area of the first branchial arch segment.** Klin. Monatsbl. f. Augenh. **135**:378-388, 1959.

Several syndromes seem genetically related to the classical mandibulofacial dysplasia of Franceschetti. Goldenhar reported malformations which he called the oculo-auricular syndrome. This consists of subconjunctival fiberodermoids near the limbus, deformities of the auricle, and a preauricular fistula. Nager and De Reynier described the mandibular syndrome in which the ocular malformations are less pronounced. Hallermann and Streiff discussed a syndrome which is characterized by bilateral microphthalmia and congenital cataracts. The facial anomalies are similar to but not identical with those of Franceschetti's syndrome. The patients

have a characteristic bird face. Ullrich and Fremerey-Dohna described a patient with a Hallermann-Streiff syndrome which had, in addition, dyscephaly, nanism, skin atrophies, and anomalies of the cranial sutures. Weyers and Thier noted multiple malformations of the vertebrae and ribs associated with oculofacial syndromes.

Most authors assume that maldevelopment of the first branchial arch segment on an endogenous basis is the etiologic factor for these deformities. It is of great interest, however, that oxygen depletion of rabbits on the eighth day of pregnancy can cause identical or similar malformations in the young animal. (10 figures, 1 table, 15 references)

Gunter K. von Noorden.

20
HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Barraquer Cerero, Tomas. **The need for applying logic to ophthalmology and medicine in general.** Arch. Soc. oftal. hispano-am. **19**:168-375, May, 1959.

The principles of logic are reviewed, their application to medicine discussed, and the description of iritis used to exemplify the application of logic in ophthalmology. The descriptions of Axenfeld and Duke-Elder are examples of description without logic, and that of Fuchs is done with reference to logic. The earliest reference the author found on the relation of logic to medicine was that of Paycha in 1957. (16 references)

Ray K. Daily.

Ophthalmic Miniature

A laboring man, below middle stature, 42 years of age, but much older in appearance, came to me on the 20th of May, 1855, complaining of dimness of sight in his left eye, attended with pain deep at the back of the eye, and beating and throbbing in the eye and over the eyebrow, extending to the temple and ear, and much increased on stooping. The globe was prominent, and protruded towards the temple; the conjunctiva was vascular, chiefly over the rectus internus muscle. No tumor of any kind could be felt with the finger. The appearance of the eyelids was natural.

Upon making firm pressure over the trunk of the left carotid artery, the globe returned to its proper position; the man smiled, and said that his sight was clear, and that all pain and throbbing had ceased. Upon relaxing pressure, the globe again protruded, and other symptoms returned.

Mr. E. L. Hussey (of Oxford),
Royal London Ophth. Hosp. Reports, **2**:127, 1859.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.

411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

ANNOUNCEMENTS

NEW YORK EYE AND EAR MEETING

The annual spring meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 4th to 7th at the infirmary.

The meeting will be oriented toward "Pediatric ophthalmology." Symposia will be offered on: "Nystagmus as a pediatric problem," "Ocular complications attending infectious diseases of childhood," "Surgical correction of congenital deformities of eye and adnexa," "Glaucoma and cataracts in pediatrics," and "Office management of the pediatric eye patient." There will also be a closed-circuit television demonstration of surgical operations.

In addition, courses will be offered in the following subjects: reading problems, pediatric anesthesiology, pleoptics, congenital syndromes, pediatric ophthalmic therapeutics, ocular neoplasms and their treatment, pediatric neuro-ophthalmologic syndromes, vertical eye muscles, pediatric ocular allergy, lacrimal sacs, and fundus lesions of childhood.

Additional information may be obtained by writing to Dr. John R. Finlay, secretary, Alumni Association, 218 Second Avenue, New York 3, New York.

SPRING POSTGRADUATE COURSES

The following postgraduate courses will be offered in the spring of 1960 at the New York Eye and Ear Infirmary.

1. Ophthalmic plastic surgery. Given under the direction of Drs. Wendell L. Hughes, J. Gordon Cole, Byron C. Smith, et al. April 25th-May 14th. Limited to eight. Fee: $250.00

2. Histopathology of the eye. Given under the direction of Drs. Bernard Roberts and Lawrence Samuels. April 25th-30th. Limited to 15. Fee: $100.00

3. Practical aspects of perimetry. Given under the direction of Dr. Mortimer Cholst. April 25th-30th. Limited to 15. Fee: $40.00

4. Ocular surgery. Includes lacrimal sac surgery, retinal detachment, cataracts, enucleation and evisceration, keratectomies and keratoplasties, anomalies of extraocular muscles, including ptosis, glaucoma, and orbitotomy. May 16th-21st. Limited to 20. Fee: $200.00

For registration, please apply to: Mrs. Tamar Weber, registrar, Institute of Ophthalmology of the Americas, New York Eye and Ear Infirmary, 218 Second Avenue, New York 3, New York.

WASHINGTON HOSPITAL CENTER

Retinal diseases will be the subject for the March 12th, 19th and 26th lectures sponsored by the Department of Ophthalmology of the Washington Hospital Center. Dr. George N. Wise will discuss "Arterio- and arteriolosclerosis," "Vascular retinopathies," and "Thrombosis and embolism," on March 12th. On March 19th, Dr. John W. McTigue will speak on "Eales' disease, Coats' disease and hemangiomatosis retinae"; Dr. Henry L. Bastien, "Senile degenerations and treatment"; and Dr. Harry M. McAllister, "Lesions at posterior pole and differential diagnosis." On March 26th, Dr. Marshall M. Parks will discuss "Infantile and juvenile macular degenerations"; Dr. Nicholas G. Pistolas, "Retrolental fibroplasia and anoxia"; Dr. Dan G. Albert, "Retinoblastoma."

Dr. Alson E. Braley, Iowa City, Iowa, will be the visiting lecturer on April 2nd when the conjunctiva will be the subject for discussion. Dr. Braley will speak on "Bacterial and viral inflammations," "Allergic inflammations," and "Degenerations, cysts and tumors."

The cornea will be the subject of the lectures on April 9th and 16th. On April 9th, Dr. L. Conner Moss will speak on "Superficial keratitis"; Dr. Thomas E. Haggerty on "Deep keratitis"; and Dr. Ben S. Fine on "Degenerations and dystrophies." On April 16th, Dr. Robert Day will discuss "Penetrating keratoplasty," and Dr. John H. King, Jr., "Lamellar keratoplasty."

COURSE ON LIGHT COAGULATION

The next introductory course on light coagulation to be given in English will be held at Essen, Germany, from April 9th to 14th. A second course to be given in English will be held from November 29th to December 3rd. For further information write to: Prof. Dr. G. Meyer-Schwickerath, Hufelandstrasse 55, Essen.

OREGON POSTGRADUATE CONVENTION

The Oregon Academy of Ophthalmology and Otolaryngology and the University of Oregon Medical School announce the 19th annual ophthalmology and otolaryngology postgraduate convention to be held March 10th to 15th. Guest speakers for ophthalmology will be Dr. Gordon Bruce, New York, and Dr. Phillips Thygeson, San Jose, California. For further information write to: Dr. Robert E. Fischer, 1216 S. W. Yamhill Street, Portland 5, Oregon.

Oxford congress

The 45th annual meeting of the Oxford Ophthalmological Congress will assemble at Balliol College, Oxford, on Sunday evening, July 10th, and the meetings will be held Monday, Tuesday and Wednesday, July 11th, 12th and 13th. The Doyne Memorial Lecture will be delivered on Tuesday morning by Prof. Norman Ashton, London, who will speak on "The exit pathway of the aqueous." For further information write to the honorary secretary and treasurer, Ian C. Fraser, 21 Dogpole, Shrewsbury, England.

Societies

Society of allergy officers

Officers for the 1959-60 season of the American Society of Ophthalmologic and Otolaryngologic Allergy are: President, Dr. Walter E. Owen, Peoria, Illinois; president-elect, Dr. Leland H. Prewitt, Ottumwa, Iowa; vice president, Dr. D. A. Skinner, Newark, Ohio; secretary-treasurer, Dr. D. S. De Stio, Pittsburgh. Members of the council are: Drs. Victor R. Alfaro, Washington, D.C.; Jack Robert Anderson, New Orleans; Bernard M. Barrett, Pensacola, Florida; William B. Barry, Kansas City, Missouri; Kenneth L. Craft, Indianapolis; French K. Hansel, Saint Louis; Francis A. O'Toole, Clinton, Massachusetts; Frederick H. Theodore, New York; and Sam H. Sanders, Memphis.

Madrid societies

Newly elected officers of the Madrid Ophthalmological Society are: President, Dr. Marin-Amat; vice president, Dr. Tena Ibarra; treasurer, Dr. Aguilar Muñoz; and secretary, Dr. Arjona Santos.

West Virginia meeting

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, West Virginia, from April 10th to 12th.

Among the guest speakers will be: Dr. Harold G. Scheie, Philadelphia; Dr. Charles E. Iliff, Baltimore; and Dr. Oscar T. Becker, Chicago. For any additional information please contact the secretary, Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington 1, West Virginia.

British Columbia Conference

The 1960 British Columbia Oto-Ophthalmological Conference will be held in Vancouver, British Columbia, on May 11th, 12th, and 13th. Guest speakers will be Drs. LeRoy A. Schall and Charles L. Schepens of Boston. There will be lectures, round-table discussions, and film sessions, and a social program for the ladies.

Those interested in attending may obtain further information from Dr. G. A. Badger, 925 West Georgia Street, Vancouver 1, British Columbia.

Holland Eye Hospitals Jubilee

Dr. Roland I. Pritikin of Rockford, Illinois (Col. MC, USAR, Res. & Dev. SGO-DA), gave one of the principal addresses at the golden jubilee of the founding of the Henry Holland Eye Hospital at Shikarpur, Sind, West Pakistan. Dr. Pritikin is the president of the Henry Holland Hospitals Alumni Association and Henry Holland Mission Hospitals Fund. This association and fund is made up of the American eye surgeons who have been coming to Shikarpur to do eye surgery since 1911. This year Dr. Pritikin is accompanied by Dr. Kenneth Somers, Washington, D.C. (Col. MC, USA, assistant chief Eye Clinic, Walter Reed Army Hospital). They are making a study of zonulolysis in cataract surgery. Another feature of the ceremonies was the presentation of an ambulance to the hospitals. This will be used by R. W. B. Holland to bring ophthalmologic services to the remote villages on the Pakistan-Afghanistan frontier. This ambulance is a gift of the Henry Holland Hospitals Alumni Association and Fund to the Henry Holland Hospitals. In his address, Dr. Pritikin thanked Dr. Victor E. Lepisto of Hancock, Michigan, for his untiring work for the Fund, and Dr. Derrick Vail, Chicago, Illinois. Dr. Lepisto is the secretary-treasurer, and Dr. Vail, the honorary chairman, Board of Directors.

Miscellaneous

Emory pathology course

Guest speakers on the program for the recent postgraduate course on "Applied ophthalmic pathology." sponsored by the Department of Ophthalmology of the Emory University School of Medicine, Atlanta, Georgia, were: Dr. Lorenz E. Zimmerman, Washington, D.C., Dr. J. A. C. Wadsworth, New York, Dr. T. E. Sanders, Saint Louis, and Dr. John T. Godwin, assistant professor of clinical pathology, Emory University.

Dr. Zimmerman spoke on "Etiology of granulomatous uveitis," "Ocular fungus infections," and "Pathology, prognosis and management of tumors of the lacrimal gland." Dr. Wadsworth discussed "Complications of cataract surgery," "Vitreous changes and retinal detachment," and "The pathologic basis for lesions of the fundus."

The subjects of Dr. Sanders' lectures were "Pathologic mechanisms of primary and secondary glaucoma," "Retinoblastoma and pseudoglioma," and "Pathologic background of retinal vascular lesions." Dr. Godwin spoke on "Pathologic anatomy of the monocular components of various ocular syndromes," "Histopathology of tumors of the orbit," and "Differential diagnosis of tumors of the eyelids." Panel discussions on "Anatomy and pathology of the vitreous and significance of changes," and "Differential diagnosis, prognosis and treatment of epibulbar and intraocular tumors," were also held.

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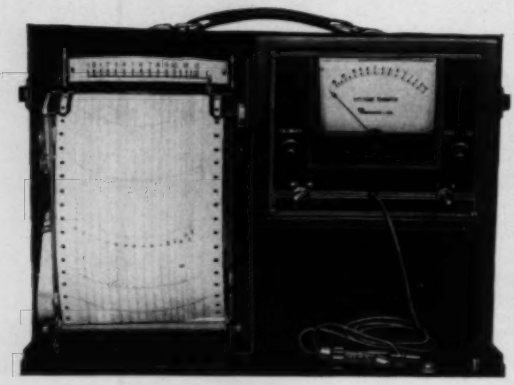

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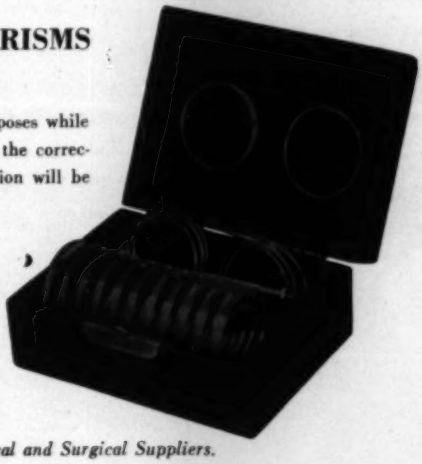

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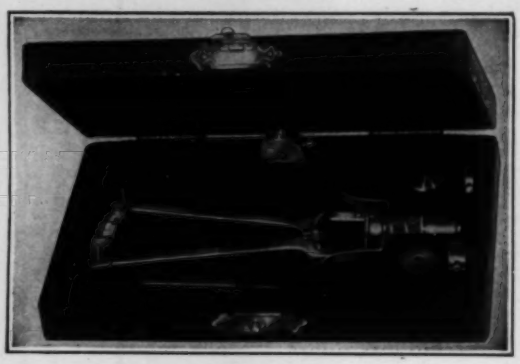

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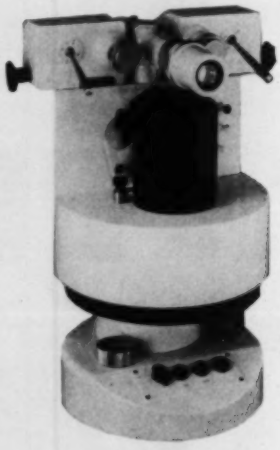

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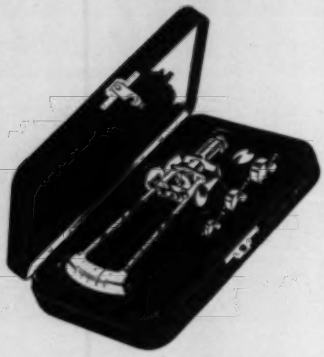

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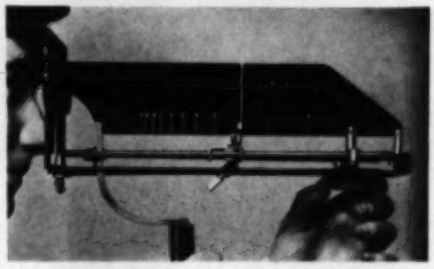

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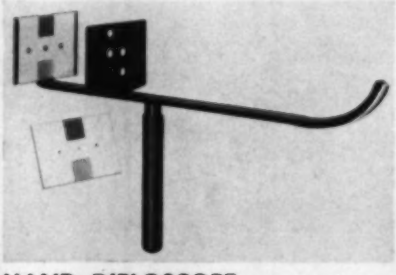

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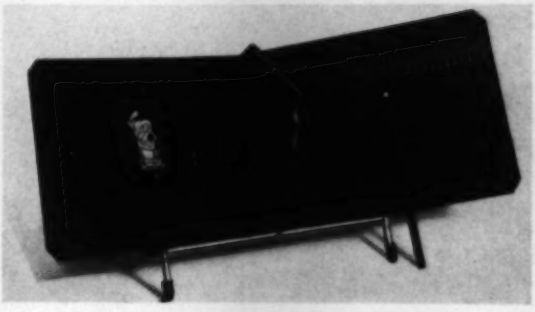

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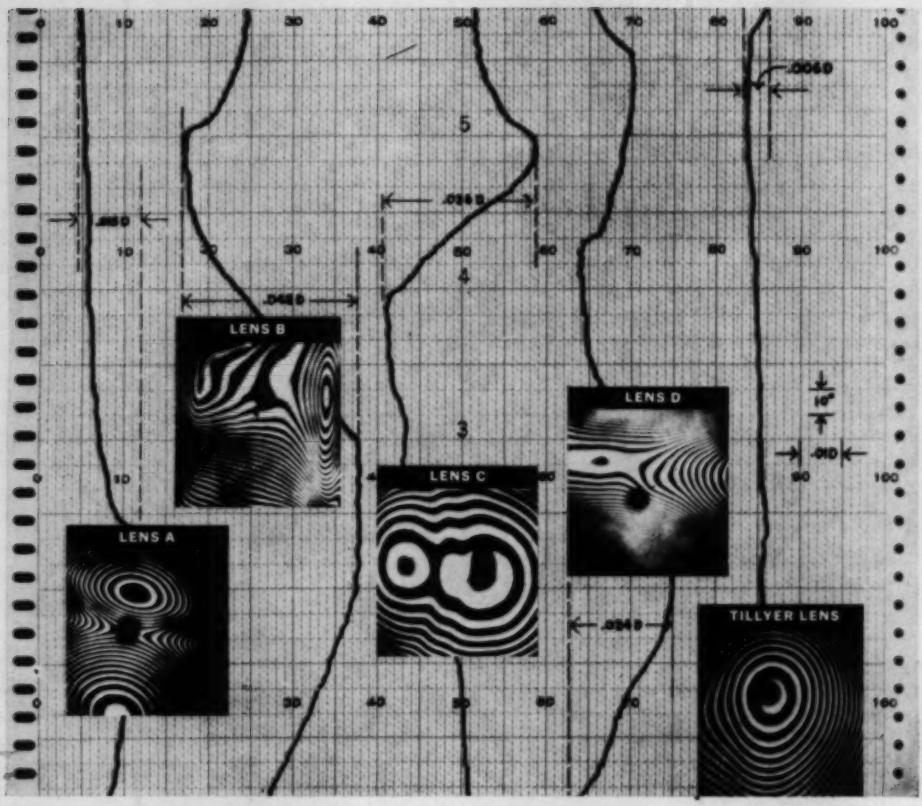

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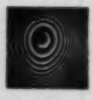

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